VARIATIONS IN OLFACTORY PARAMETERS IN DIFFERENT OLFACTORY DISORDERS - A RETROSPECTIVE STUDY

A dissertation submitted to the Tamil Nadu Dr. M. G. R. Medical University, Chennai in partial fulfilment of the requirement for the MS Otorhinolaryngology (Branch IV) degree examination to be held in May 2022.

VARIATIONS IN OLFACTORY PARAMETERS IN DIFFERENT OLFACTORY DISORDERS - A RETROSPECTIVE STUDY

Dissertation submitted to the

THE TAMIL NADU DR. MGR MEDICAL UNIVERSITY, CHENNAI

In partial fulfilment of the requirements for the degree of

MASTER OF SURGERY

IN

OTORHINOLARYNGOLOGY

By

BERENICE STELLA. B

Register number: 221914351

DEPARTMENT OF OTORHINOLARYNGOLOGY

CHRISTIAN MEDICAL COLLEGE

VELLORE

MAY 2022

CERTIFICATE

This is to certify that "**Variations in olfactory parameters in different olfactory disorders - A retrospective study**" is the bona-fide work of Dr. Berenice Stella. B under my supervision in the Department of Otorhinolaryngology, Christian Medical College Vellore in partial fulfilment of the requirements for the M.S ENT Examination Branch IV of the Tamil Nadu Dr. M.G.R Medical University to be held in May 2022 and no part thereof has been submitted for any other degree.

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DECLARATION

I, Berenice Stella. B, do hereby declare that the dissertation titled **"Variations in olfactory parameters in different olfactory disorders - A retrospective study**" is a genuine record of research done by me under the supervision and guidance of Dr. Lalee Varghese, Professor, ENT, Christian Medical College, Vellore and has not previously formed the basis of award of any degree, diploma, fellowship or other similar title of any university or institution.

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I also would like to thank my statistician, Dr. Grace Rebekah. J for her hard work and time spent to help me analyse my data.

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I also feel extremely blessed to have my husband Dr. Geoffery Daniel Kirubakaran, and my father Mr. Brabahar Solomon, who were there every step of the journey, constantly supporting me, helping me keep timelines and always cheering me on.

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Dr. Berenice Stella B,(Emp. No. 80269) PG Registrar, Otorhinolaryngology, Dr. Lalee Varghese, ENT Unit -3, Dr. Regi Kurien (Employment number: 31688), ENT Unit -3.

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Dear Dr. Berenice Stella B,

I enclose the following documents:-

1. Institutional Review Board approval 2. Agreement

Could you please sign the agreement and send it to Dr. Suceena Alexander, Addl. Vice Principal (Research), so that the grant money can be released.

With best wishes,

Dr. Suceena Alexander
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Dear Dr. Berenice Stella B,

The Institutional Review Board **(Blue**, Research and Ethics Committee) of the Christian Medical College, Vellore, reviewed and discussed your project titled "Variations in olfactory parameters in different olfactory disorders – A retrospective study" on September 02, 2020.

The Committee reviewed the following documents:

1. IRB Application Form
2. Proforma
3. HOU Permission Letter
4. Cvs. of Drs. Regi Kurien, Lalee Varghese, Berenice Stella.
5. No.of Documents 1 – 4.

The following Institutional Review Board (Blue, Research & Ethics Committee) members were present at the meeting held on September 02, 2020 in the New IRB Room, Christian Medical College, Vellore 632 004.

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Dr. Premila Abraham	M.Sc. Ph.D	Professor, Department of Biochemistry, CMC, Vellore	Internal Clinician

We approve the project to be conducted as presented.

Kindly provide the total number of patients enrolled in your study and the total number of Withdrawals for the study entitled: "Variations in olfactory parameters in different olfactory disorders – A retrospective study" on a monthly basis. Please send copies of this to the Research Office (research@cmcvellore.ac.in).

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The Institutional Ethics Committee expects to be informed about the progress of the project, Any **adverse events** occurring in the course of the project, any **amendments in the protocol and the patient information / informed consent**. On completion of the study you are expected to submit a copy of the **final report**. Respective forms can be downloaded from the following link: http://172.16.11.136/Research/IRB_Polices.html in the CMC Intranet and in the CMC website link address: http://www.cmch-vellore.edu/static/research/Index.html.

Fluid Grant Allocation:

A sum of 8,000/- INR (Rupees Eight Thousand Only) will be granted for 12 Month.

Yours sincerely,

Dr. Suceena Alexander
Secretary (Ethics Committee)
Institutional Review Board

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Secretary - (Ethics Committee)
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ABSTRACT

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AIM

The aim of this study is to analyse the variations in *n*-butanol threshold and odour identification scores of the CCCRC test (Connecticut Chemosensory Clinical Research Centre) in patients with various olfactory disorders.

STUDY DESIGN

This was a retrospective observational cohort study.

MATERIALS AND METHODS

Patients for this study were recruited retrospectively from CMC database. All patients who were diagnosed with olfactory dysfunction, between January 2018 and March 2020, based on CCCRC olfaction testing were selected retrospectively after applying the inclusion and exclusion criteria. After patient selection, details of each patient including demography, presenting symptoms, duration of symptoms, clinical examination and olfaction testing details were recorded and analysed.

RESULTS AND CONCLUSION

At the end of the study, the *n*-butanol threshold scores and the odour identification scores were analysed for variations in each type of the olfactory dysfunction, comorbidities and nasal diseases. We found that the odour identification component of the CCCRC test plays a major role in determining the composite score and thereby the type of olfactory dysfunction. Among the

seven odours tested for odour identification, pepper was the most affected smell among most of the nasal diseases and comorbidity groups.

INTRODUCTION

INTRODUCTION

The nose acts as a major gateway for respiration, but also has other vital functions that are essential for enhancing the quality of life. The functions of the nose are respiration, protection of lower airways, vocal resonance, humidification of inspired air and olfaction.

It plays a vital role in filtering and purifying the inspired air and also alters the temperature and humidity of the air before it reaches the lungs. The mucosal lining of the nose has enormous mucous secreting glands, which secretes a mucous blanket which clears the inspired bacteria, virus and dust and takes it to the nasopharynx, thereby protecting the lower airways.

The olfactory function of the nose is vital in detecting environment chemicals. Humans depend on a well-functioning sense of smell to distinguish hazardous smells like smoke, gas leaks and chemicals. The smell also predicts the general mood of a person. The inability to sense smell results in significant psychological disruption, social vulnerability and victimization. Therefore, it significantly contributes to the quality of daily life.

When sense of smell combines with taste and somatosensory stimuli, the various flavours of foods are identified and thereby enjoyed. This also aids the process of digestion by triggering normal gastrointestinal secretions.

The olfactory region is found in the roof of the nose in humans. It has a specialized epithelium containing sensory neurons carrying olfactory sensation to the brain. They have the ability to regenerate over period of time (1).

The sense of smell is altered by various nasal diseases, of which chronic rhinosinusitis, allergic rhinitis, atrophic rhinitis are most commonly seen. Other causes for acute loss of smell include trauma to the olfactory region or following transnasal approach surgeries.

Olfactory dysfunction can be caused by different mechanisms. It could be a conductive loss due to obstruction of nasal passages, sensorineural loss due to damage to olfactory neuroepithelium or due to a central olfactory neural loss.

The inability to perceive smell is termed as anosmia, while the reduced perception to smell is hyposmia. Threshold depends on the chemical nature of stimuli and the level of inhibitory activity from the higher centres. The threshold of perception is lower than identification that is a smell is sensed before it is recognized.

In this study, the pattern of smell loss in different olfactory disorders using CCCRC olfaction testing has been studied and analyzed. The relationship between systemic illness like diabetes mellitus, hypertension, dyslipidemia, bronchial asthma and hypothyroidism with loss of smell has also been addressed.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

EMBRYOLOGY

The nose is developed from mesenchymal processes that are present around the primitive mouth. This process begins by the fourth week of intrauterine life. Collections of neural crest cells undergo proliferation and form the nasal placodes. These nasal placodes then sink in the centre to transform into nasal pits, which deepens to form the nasal sac. The adjacent mesodermal cells increase rapidly in number and gives rise to the medial and lateral nasal prominences of the frontonasal process. These processes surround the nasal pit and sac which together form the nares.

The maxillary processes enlarge anteriorly to join the medial nasal prominences and combines medially with the frontonasal process forming separate nasal cavities by closing off the nasal pits. The bucconasal membrane separates the primitive nasal cavity and mouth at first. This membrane thins out in due course and finally breaks down to form the choanae.

The frontal and maxillary processes fuses to form the lateral two thirds of the upper lip, superior alveolar ridges and palatal shelves. The medial nasal prominences combine with the maxillary process to develop into the philtrum and medial crus of the lower lateral cartilages. The nasal bones, upper lateral cartilages and lateral crus of the lower lateral cartilages are formed by the lateral nasal prominences.

The posterior midline growth of the frontonasal process into the root of the oral cavity develops the nasal septum which further extends posteriorly to form the opening of the Rathke's pouch. The primitive or primary palate is formed, anteriorly by the fusion of the maxillary and frontonasal processes. The palatal processes which are

derived from the lateral maxillary mesoderm, begins to grow medially towards each other and the nasal septum with the enlargement of the nasal cavity. Initially, the palatal processes are vertically oriented and lateral to the tongue. During the development of the jaw and oral cavity the palatal processes begins to migrate medially towards the midline and fuse. This fusion initiates at the posterior margin of the primitive palate and then progresses from anterior to posterior to form the secondary palate.

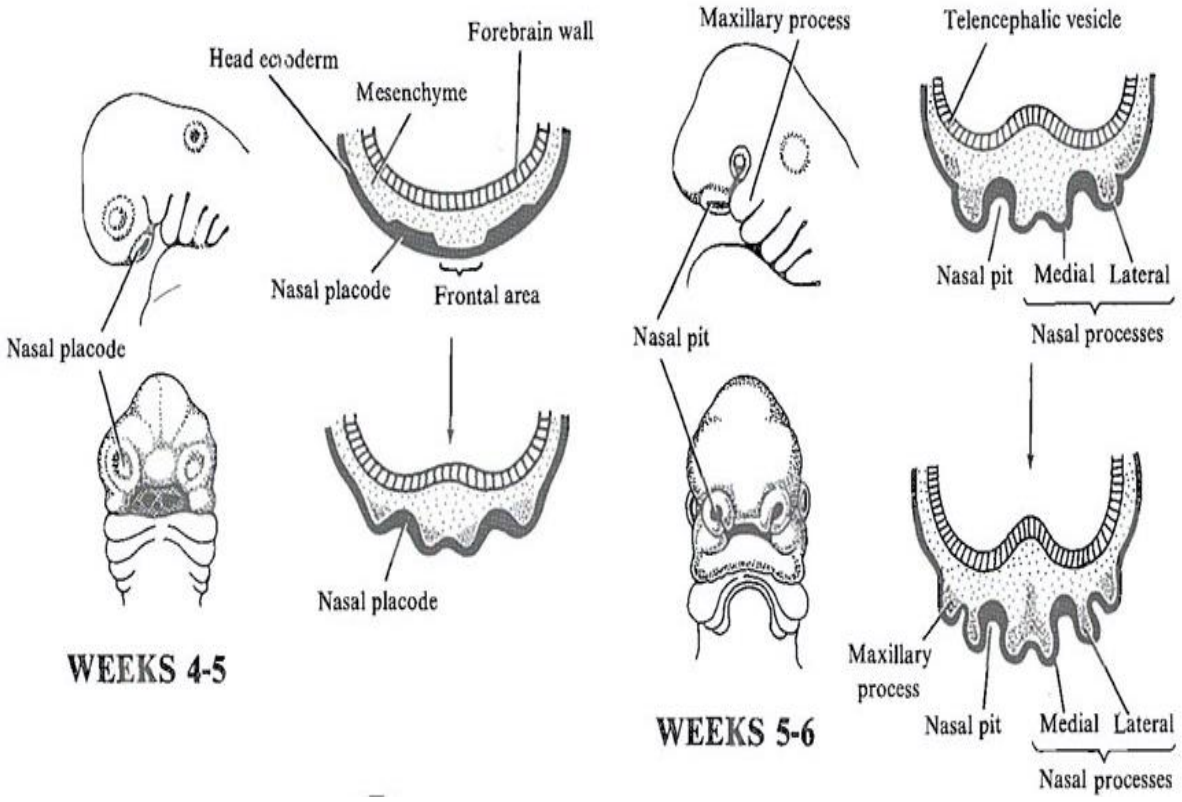


Figure 1 - Development of nasal cavity

(Source -Review of Medical embryology Book by Ben Pansky,

Chapter 57. Development of The Nasal Cavities)

ANATOMY

The human nose has two nasal passages that serve as channels for both respiration and olfaction. It helps to warm and humidify the inspired air while eliminating most of the airborne pathogens and environment pollutants, most of which are toxic to the olfactory system.

EXTERNAL NOSE

The external nose has an osteocartilaginous framework and is further made of muscles and skin. The external nose is bony in its upper one third and cartilaginous for the lower two thirds. The bony part is made up of two nasal bones which articulates in the midline and rest on the upper part of the nasal process of the frontal bones and supported by the frontal processes of the maxillae on either side.

The cartilaginous part is made up of four cartilages. The upper lateral cartilages bridge the undersurface of the nasal bones above, to the alar cartilages below. They are connected with each other in the midline and anteriorly with the upper border of the septal cartilage. The lower lateral cartilages or alar cartilages are two in number which are U-shaped. Each alar cartilage has a lateral crus which constitutes the ala and a medial crus which runs in the columella. The lesser alar or sesamoid cartilages are two or more in number which are situated above and lateral to the alar cartilages. The dorsum of the nose is supported by the septal cartilage.

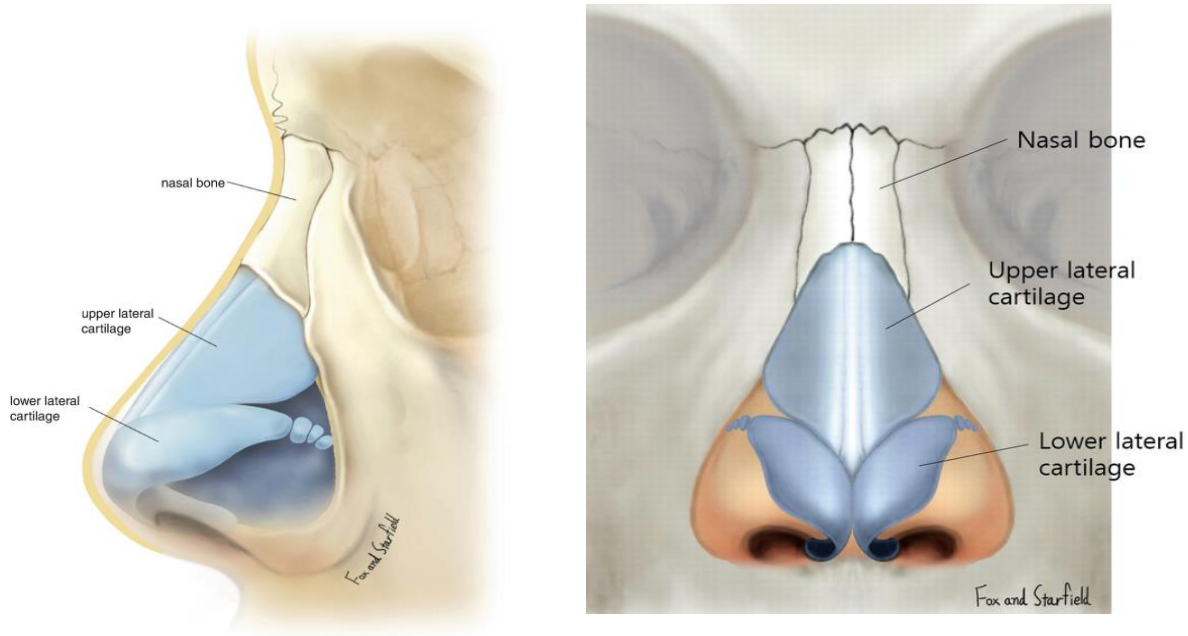


Figure 2 - Osteo-cartilaginous framework of nose

(Source -Atlas of Asian Rhinoplasty pp 1-65 - Anatomy and physiology of nose)

There are four muscles which cover the osteocartilaginous framework of nose which are responsible for the movements of the nasal tip, ala and the skin overlying them. They are the procerus, nasalis (transverse and alar parts), levator labii superioris alaeque nasi, anterior and posterior dilator nares and depressor septi.

The skin is thin and freely mobile over the nasal bones and upper lateral cartilages but is thick and adherent over the alar cartilages which has numerous sebaceous glands.

NASAL CAVITY

The nasal cavity from the anterior nares to nasopharynx is lined by ciliated columnar epithelium and by olfactory epithelium in the superior part. The midline

septum separates the nasal cavities into two with each comprising of a roof, floor, lateral and medial walls, and anterior and posterior apertures. Each nasal cavity communicates with the exterior through the nostril and posteriorly with the nasopharynx via the posterior nasal aperture or the choana. Each nasal cavity consists of the vestibule and a nasal cavity proper. The vestibule in the external nose has coarse hairs which helps to keep away the dirt particles from the inspired air from the upper airway.

The roof is narrow arched and it lies below the anterior cranial fossa. The nasal cavity is built by the nasal cartilages, nasal and frontal bones, the cribriform plate of the ethmoid and the body of the sphenoid from anterior to posterior. The floor is horizontal and it also participates in the formation of the roof of the oral cavity mainly by the palatine process of the maxilla and the horizontal plate of the palatine bone.

NASAL SEPTUM

The nasal septum separates the nasal cavity into two, gives support to the nasal dorsum and also maintains the tip of the nose. It consists of a bony part, cartilaginous part and membranous part. The bony part of the septum is formed by the perpendicular plate of ethmoid, vomer, crest of maxilla and palatine bone. The cartilaginous part of the septum is made by the quadrangular cartilage. This cartilage is continuous with the upper lateral cartilages towards the nasal bridge. The membranous part is formed by connective tissue present between the columella and the caudal portion of quadrangular cartilage.

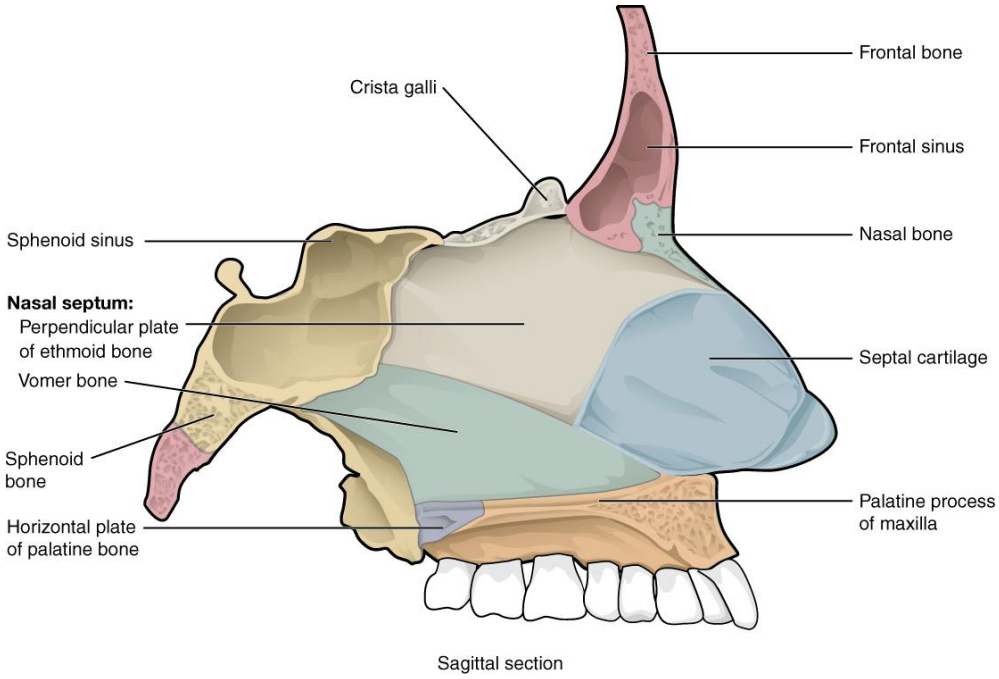


Figure 3 - Anatomy of nasal septum

(Source - Anatomy and physiology Connexions Web site.

http://cnx.org/content/col11496/1.6/, Jun 19, 2013)

LATERAL WALL OF NOSE

The lateral wall of nasal cavity is closely related to the orbit, the ethmoid and maxillary sinuses. There are three horizontal bony projections, the superior, middle and inferior nasal conchae (turbinates) which greatly increase the surface area of the lateral wall. The largest of them is the inferior concha which is an independent bone and it is situated about 1 cm above the floor of the nose. Underneath each concha is a meatus, and just above the superior concha there is a triangular fossa, sphenoethmoidal recess into which the sphenoid sinus opens.

The nasolacrimal duct opens into the inferior meatus, whereas the posterior ethmoidal air cells open into the superior meatus. The middle meatus has a rounded

elevation, the bulla ethmoidalis. A deep semicircular groove just below the bulla is called as the hiatus semilunaris, which receives the openings of the frontal, anterior ethmoidal and maxillary air sinuses. There is a short passage at the anterior end of the hiatus called as the infundibulum (2).

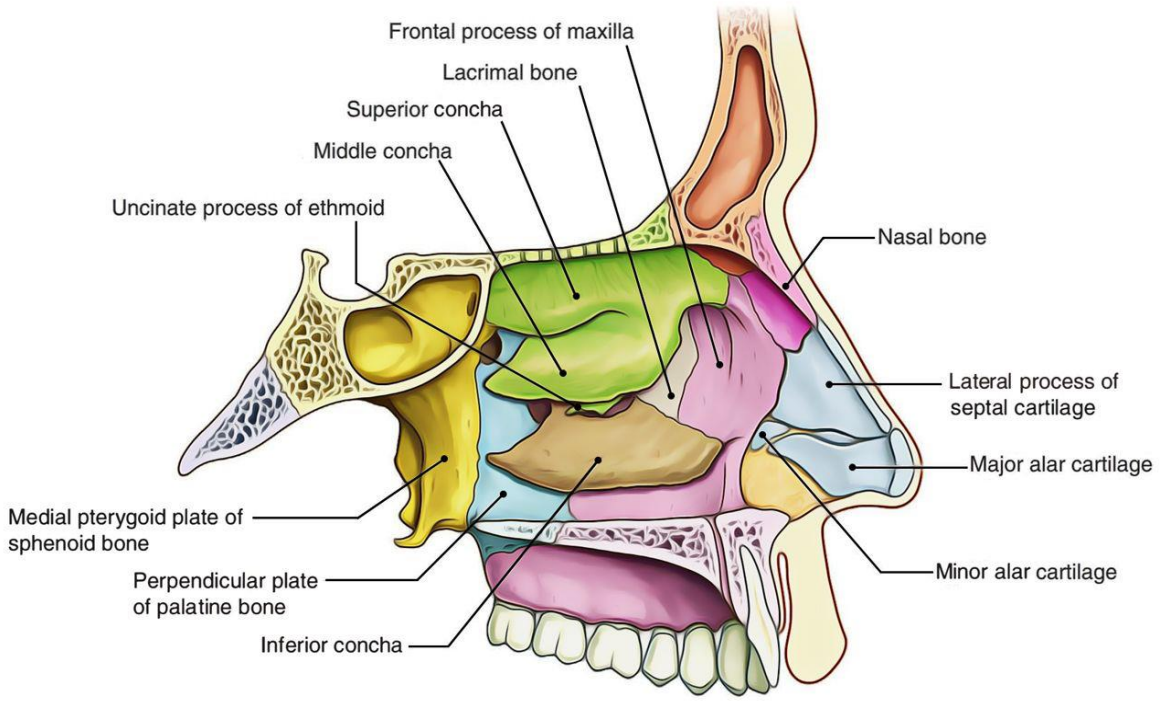


Figure 4 - Anatomy of lateral wall of nose

(Source - Earth's lab - Anatomy of nasal cavity)

BLOOD SUPPLY OF NOSE

The nose is primarily supplied by the branches of the external and internal carotid arteries namely maxillary, facial and ophthalmic arteries. These branches anastomose within the nose and on the nasal mucosa. The anterior and posterior ethmoidal arteries are branches of ophthalmic artery and supplies the roof of nose along with ethmoid and frontal sinuses.

The sphenopalatine artery is a branch of maxillary artery which supplies the mucosa of the meatuses, turbinates and posteroinferior part of the septum. This artery divides into posterior septal and posterior lateral nasal branches before entering the nasal cavity. The Little's area or Kiesselbach's plexus is the region of anastomosis on the anteroinferior part of the septum by the sphenopalatine, anterior ethmoid, greater palatine and septal branch of superior labial arteries.

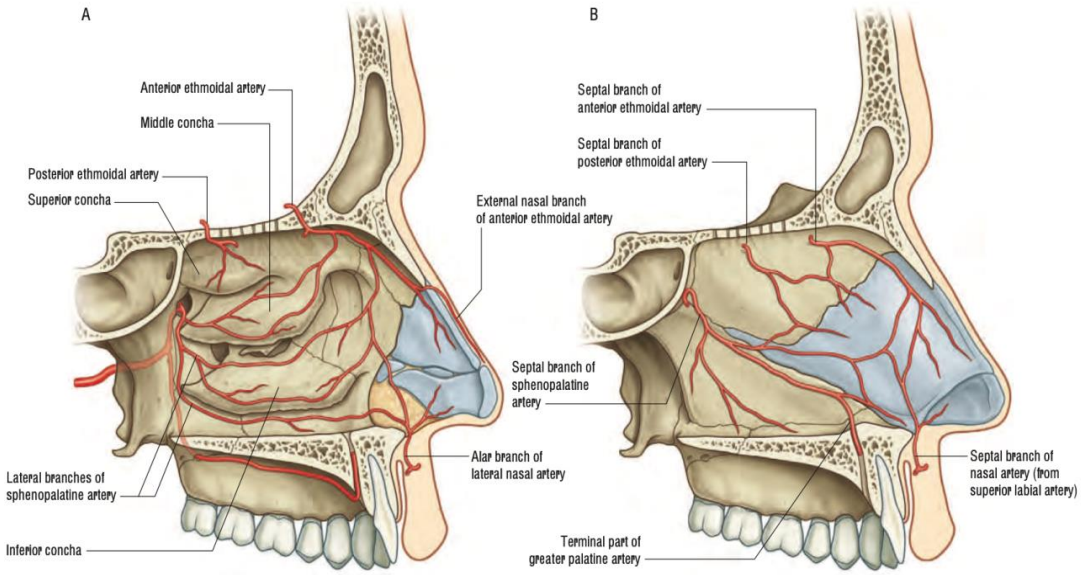


Figure 5 - Blood supply of A) lateral wall of nose, B) nasal septum

(From DrakRL, Vogl AW, Mitchell A (eds), Gray's Anatomy for Students, 2nd ed, Elsevier, Churchill Livingstone. Copyright 2010)

The veins draining the posterior part of the nose drains into the pterygoid venous plexus through the sphenopalatine vein crossing the sphenopalatine foramen. The anterior part of the nose is drained by the anterior ethmoid veins and finally drain into the facial or ophthalmic veins.

OLFACTORY EPITHELIUM

The olfactory cleft is a 1mm wide opening that is present 7cm deep to the nostril. The olfactory neuroepithelium is present in this small region of nasal mucosa measuring about approximately 2cm square situated in the upper recesses of the nasal chamber, which is lining the cribriform plate and also forms part of the superior turbinate, middle turbinate, and septum.

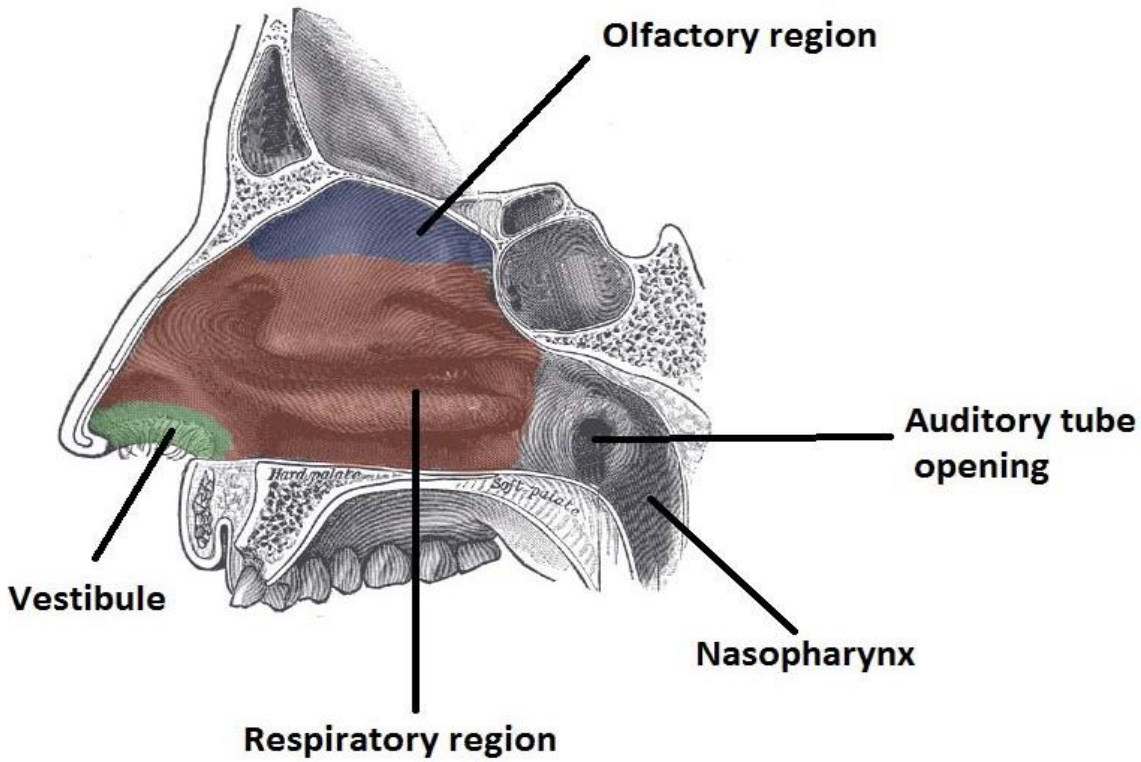


Figure 6 - Olfactory region in the nasal cavity

(Source -teachmeanatomy.info/the nasal cavity)

The major part of the inspired air entering the nose is shunted around the inferior meatus (35%) and middle meatus (50%) before reaching the olfactory neuroepithelium, which is only 10-15%. Hence minor alterations involving the nasal architecture and the

airflow can lead to considerable blockage of airflow to the olfactory regions without causing significant impairment in nasal respiratory ability.

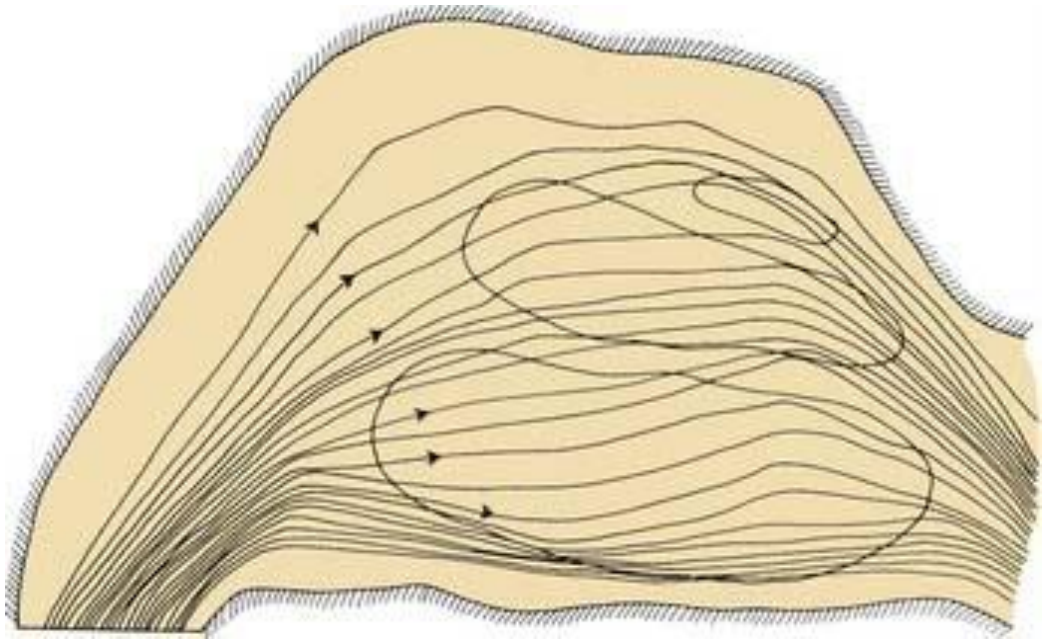


Figure 7 - Streamline patterns for resting inspiratory flow through a scale model of a healthy human adult

(Source: The biophysics of nasal airflow: Otolaryngology Clin North Am 1989:22:265)

HISTOLOGY OF OLFACTORY EPITHELIUM

Olfactory neuroepithelium is made up of pseudostratified columnar epithelium, and held by a highly vascularized lamina propria. The following are the six distinct classes of cells which are defined morphologically, biochemically, and functionally present within the neuroepithelium.

1. The bipolar sensory receptor neuron has its embryological origin from olfactory placode. Its main function is to hold out odorant receptor-containing cilia into mucus.

2. The sustentacular or supporting cell interposes between the bipolar receptor cells. It facilitates the production of mucus, transports molecules across the epithelium and also helps in detoxification and degradation of odorants.

3. The duct cell of Bowman's glands is the major contributor in production of mucus in the olfactory epithelium

4. The microvillar cell which is located at the surface of the epithelium, provides tufts of microvilli into the nasal mucus similar to the supporting cell.

5. The horizontal basal or dark cells, represents one of the two principal classes of stem cells within the basement membrane of the epithelium.

6. The globose basal or light cells, is a multipotent basal cell that is capable of giving rise to neurons and non-neuronal cells, including the horizontal basal cells.

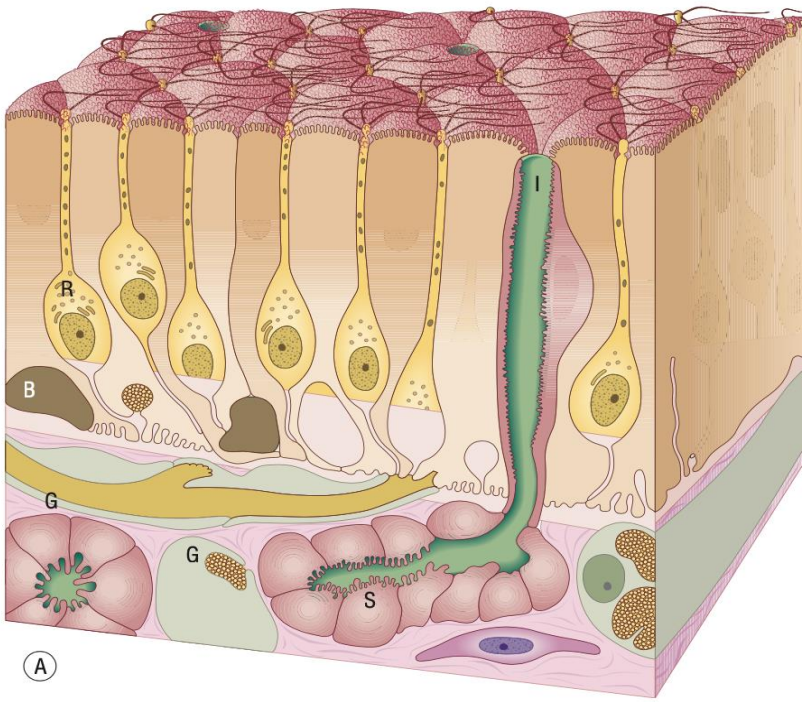


Figure 8 - Histology of olfactory epithelium

PHYSIOLOGY OF OLFACTION

1. Olfactory epithelium

The olfactory sensory neurons occupy the yellowish pigmented olfactory epithelium which is present in a specialized portion of the nasal mucosa, in the roof of the nasal cavity, close to the septum. There are about 10 to 20 million bipolar olfactory sensory neurons which are interrupted with glia like sustentacular cells and basal stem cells. Each neuron consists of a thick, short dendrite terminating as a knob with 10 to 20 cilia into the nasal cavity. These cilia are unmyelinated and measures about 2 μm in length with a diameter of 0.1μm. They possess specific receptors for odorants known as odorant receptors.

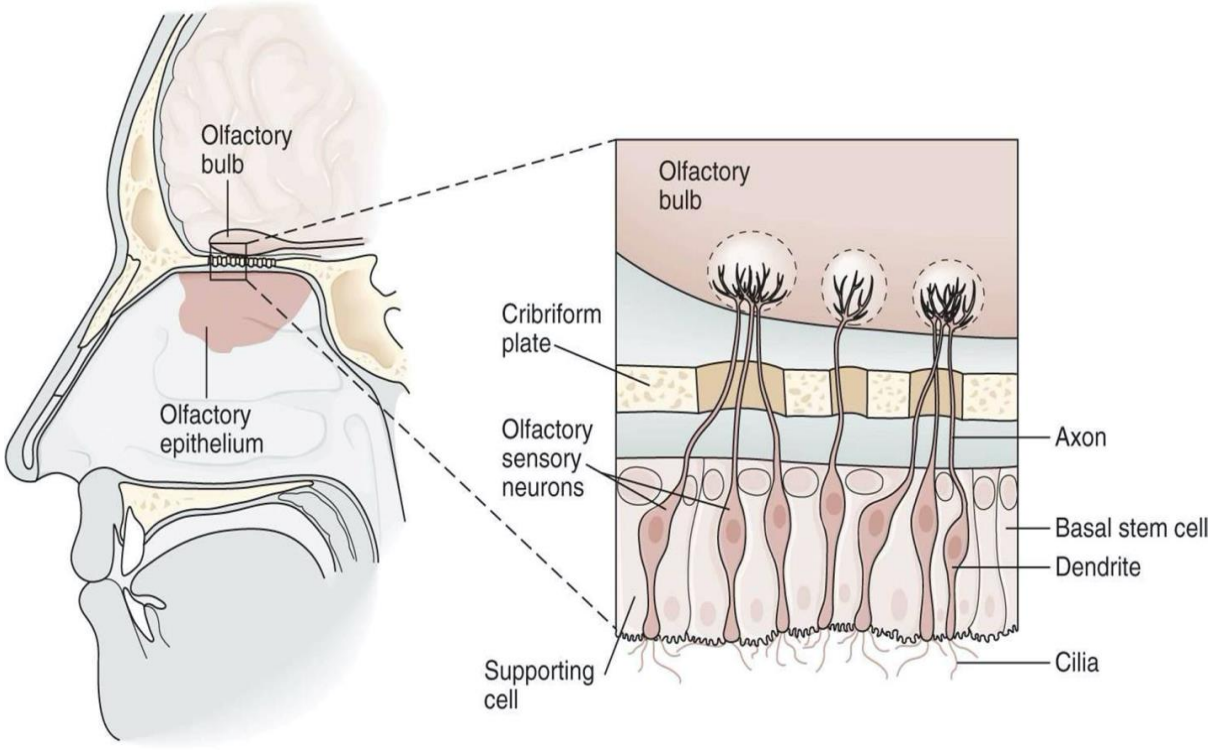


Figure 9 - Olfactory sensory neurons embedded in the olfactory epithelium

(Source - Kandel ER, Schwartz JH, Jessell TM [editors]: Principles of Neural Science, 4th ed. McGraw-Hill, 2000)

The axons of the olfactory sensory neurons enter the olfactory bulb by going through the cribriform plate of the ethmoid bone.

2. Olfactory bulbs

After reaching the olfactory bulbs, the axons of the olfactory sensory neurons join the primary dendrites of the mitral and tufted cells forming discrete synaptic units known as olfactory glomeruli. Then the mitral and tufted cells send the axons to the olfactory cortex. The olfactory bulb has periglomerular cells, which act as inhibitory neurons linking the glomeruli together. The granule cells do not possess any axons, but they are responsible for establishing reciprocal synapses with the lateral dendrites of the mitral and tufted cells. These cells excite the granule cell at the synapse by producing glutamate. Therefore, the excited granule cell inhibits the mitral and tufted cell by causing GABA release (3).

3. Olfactory cortex

The axons of the mitral and tufted cells travel posteriorly via the lateral olfactory stria and ends on the apical dendrites of the pyramidal cells at five regions of the olfactory cortex. These regions are anterior olfactory nucleus, olfactory tubercle, piriform cortex, amygdala and entorhinal cortex. The olfactory information travels from these regions to the frontal cortex, or through the thalamus to the orbitofrontal cortex.

The pathway to the orbitofrontal cortex is responsible for conscious discrimination of odours. The orbitofrontal activation is more on the right than the left side, hence there is asymmetric representation of olfaction. The pathway to the amygdala is related to the emotional responses to olfactory stimuli, whereas the entorhinal cortex pathway is involved with olfactory memories.

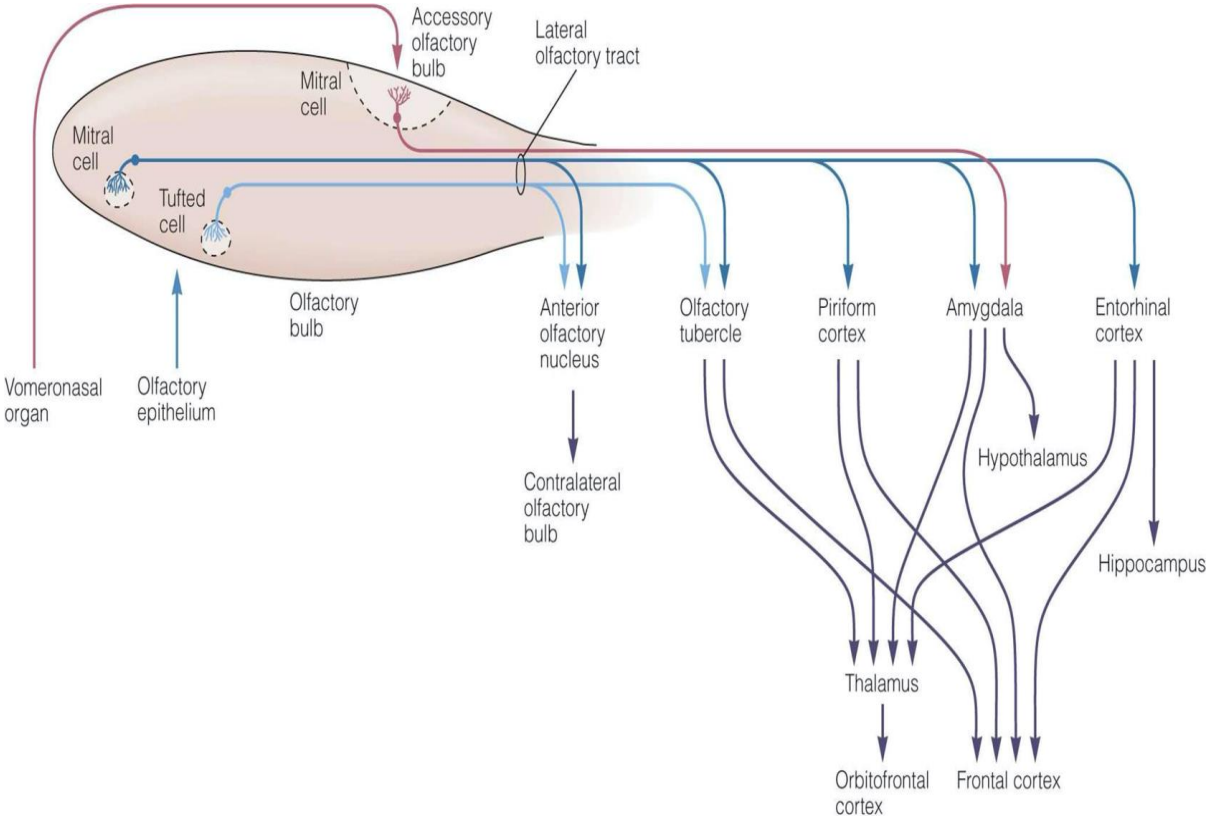


Figure 10 - Olfactory pathway

(Source - Kandel ER, Schwartz JH, Jessell TM [editors]: Principles of Neural Science, 4th ed. McGraw-Hill, 2000)

THEORIES FOR OLFACTION

1. The Vibrational theory

Malcolm Dyson in 1928 first proposed this theory which suggests that the odorant quality is based on vibrational recurrence of a molecule in the infrared range. The author conducted spectroscopic studies and derived a correlation between certain odours and the vibration frequencies of molecules.

The correlations between odour character and low-frequency molecular vibrations were limited. The other pitfall of this theory is that it failed to prove that some enantiomers produce different smells even though their vibrational spectra were identical. There was no biological mechanism identifiable as a plausible protein-based spectroscope having the ability to convert molecular vibrations into neuronal activation. Hence this theory was disregarded (4).

2. **The profile – functional theory**

Certain specific chemical groups present in a molecule are characterised by their specific odour. Beets proposed the Profile – Functional theory in 1957. He proposed that, odour is determined by two separate contributions: one from the form, size and bulk shape of the molecule; the other through its functional group or groups that define the molecular orientation at the receptor site. The ability of a functional group to orient effectively the odorant at the site, was supposed to be partly determined by its tendency to participate in hydrogen bonding interactions (5).

3. **The electrochemical theory**

According to this theory proposed by Briggs and Duncan in 1962, because the olfactory region of the nasal cavity is yellow and the respiratory epithelium is not, it comprises of carotenoids that are responsible for formation of the protein bound molecular receptors of the odorant molecules (6).

4**. Chromatography theory**

Mozell proposed this theory in 1967. Accordingly, the olfactory discrimination like chromatography depends on the ability with which the molecules of each odorant

migrate along the mucosa. The molecules of certain chemicals which possess a greater capacity to travel along the mucosa could reach the olfactory regions faster (7).

5**. The steric theory**

This theory states that air-borne molecules are identified as smell when they attach into the complementary receptor sites. Thus, the specific shape and size of the molecule is responsible for the specific odour quality. It resembles a lock and key mechanism. The molecule bonding activates the receptor coupled G – protein which in turn corresponds to the signal transduction (8).

OERP (OLFACTORY EVENT RELATED POTENTIALS)

OERPs are due to activation of areas of brain from the olfactory tracts and bulbs involving the orbitofrontal cortex with rostrum-medial regions of the temporal lobe. They are produced by electrical stimulation of the olfactory mucosa and it can be used as one of the clinical tests to detect olfactory dysfunction. The sensory input is traveling from the roof of the nasal cavity, where the olfactory neuroepithelium is located via the olfactory bulbs and cranial nerves. In the first cranial nerve, the sensations communicate with the second order neurons which are the dendrites of the mitral and tufted cells. The postsynaptic fibers arise from the second order neurons and travels to the primary olfactory areas in brain and form the olfactory tracts. The primary olfactory regions are anterior olfactory nucleus, tenia tecta, olfactory tubercle, amygdala, piriform cortex, anterior cortical amygdaloid nucleus, and entorhinal cortex (9).

OLFACTORY TRANSDUCTION

After the odorant molecule is dissolved in the olfactory mucus, a set of events ensue. The odorant-binding protein increases the concentration of the odorants in the proximity of the receptor cells as high as 1,000 to 10,000, increasing the ease of access of odorants to the olfactory receptors.

The odorant chemical information is converted to an electrical action potential by the specific interactions between odorant molecules and receptor proteins on the surface of olfactory cilia. At the receptor cell membrane, many second-messenger systems help in depolarizing the cell and starts the action potential.

The cyclic adenosine monophosphate (cAMP) and inositol phosphate (IP_3) are the prime signaling pathways channeling olfactory transduction. As the receptor is bound to an odorant, adenylate cyclase is energized by G_{olf} and converts adenosine triphosphate (ATP) into cAMP. The cAMP bound to Na, Ca ion channel permits the influx of these ions. When more channels open, in sequence, the cell depolarizes and an action potential is produced.

The peripheral olfactory receptor cells depolarization then starts a convergence of electrical information toward the olfactory bulb. The axons from olfactory receptor subtype converge if identical and synapse with mitral cells within only a few glomeruli of each olfactory bulb. A particular odorant may trigger a specific olfactory receptor types which in turn signals to specific glomeruli. Thus, a design of action similar to the designs of action of other sensory systems is created in the brain.

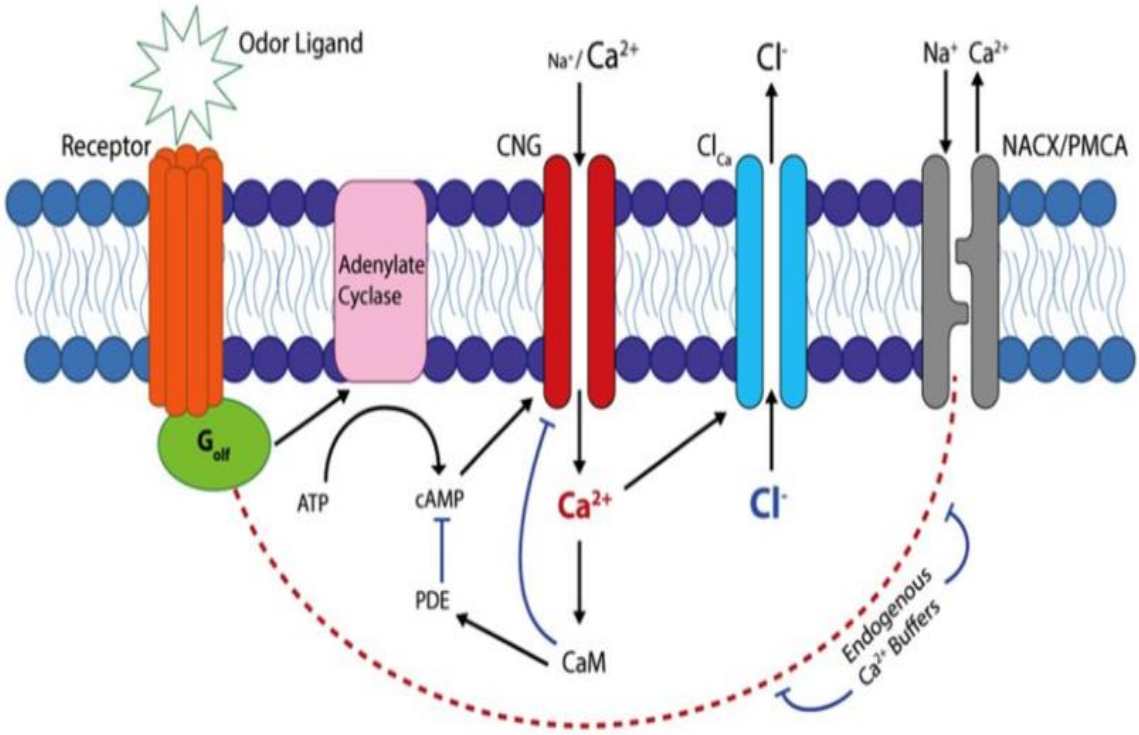


Figure 11 - Signal transduction in an odorant receptor

(Source -[*https://www.researchgate.net/figure/Transduction-in-the-cilia-of-the-olfactory-sensory-neurons-Golf-olfactory-G-protein_fig2_262980675*](https://www.researchgate.net/figure/Transduction-in-the-cilia-of-the-olfactory-sensory-neurons-Golf-olfactory-G-protein_fig2_262980675) ***)***

EFFECTS OF OLFACTION

1. Eating

The olfactory and gustatory responses provoke pleasurable moments which are transmitted to the brain. They are responsible for eating food with pleasure. Hence, if the olfaction is lost, it affects the eating considerably leading to eating disorder. The olfaction also affects the ingestive as well as digestive capacity leading to loss of weight following the loss of smell. In a systematic review done from 14 studies among anorexic

and obese patients, it was concluded that olfactory capacity is reduced in anorexia nervosa patients (10).

2. Sexuality

Studies have shown strong relationship between the olfaction and the sexual behaviour. Smell acts as a very powerful sexual stimulus. The smell from the body secretions is found pleasant in infants, whereas they are found to be repulsive or not stimulating later in life (11).

3. Psychosocial

Patients with smell dysfunction develop depressive symptoms due to low self-esteem and quality of life. In a study conducted among 22 anosmic patients in Austria, it was found that anosmic patients have reduced body related self-esteem and reduced quality of life (12).

FACTORS AFFECTING OLFACTION

1. Age

Reduced olfactory capacity is usually seen in older individuals. It is seen in half of the people between 65 to 80 years and in three quarters among people more than 80 years of age. Men have early loss of smell when compared to women. The factors that are responsible to age related loss of smell are, repeated damage to the nasal epithelium following viral infections, altered nasal engorgement, reduction of metabolizing

enzymes in the nasal mucosa, cribriform plate forming ossification, reduction in the receptor cells for odorants (13).

2. **Gender**

Women have better olfactory awareness compared to men from the early childhood, leading to better olfactory capacity in them. This is due to better neuroendocrine and cognitive abilities in women than men. Thus, women are more sensitive to low level odours compared to men in everyday life.

3**. Hormones**

There are olfactory changes associated with the different phases of menstrual cycle. It is found that the olfactory sensitivity is more pronounced during the ovulation and the luteal phase.

4. Genetic factors

Kallmann syndrome also known as hypogonadotropic hypogonadism is one the most common genetic cause of anosmia or hyposmia. The incidence ranges from 1:8000 among men to 1:40000 in women. Anosmin -1 is a protein which acts as a positive regulator of FGFR1 thereby playing a major role in neuronal cell functions. The gene coding Anosmin-1 is mutated in Kallmann syndrome (14).

5**. Smoking**

In a study conducted in Germany among 1300 patients who are smokers between 25 and 75 years, it was found that 3.6% of the participants were anosmic and 18% had significant olfactory loss (15).

TESTS FOR OLFACTION

1. UPSIT (University of Pennsylvania Smell Identification Test)

The UPSIT is a self-administered test done while in the waiting room and it takes about 10 to 15 minutes by most patients, which can be scored in less than one minute by non-medical personnel. This test contains of four booklets in many languages comprising 10 microencapsulated (‘scratch and sniff’) odorants in every piece. The test results are validated based on the percentile score of a patient’s performance in relation to the age- and sex-matched controls. The olfactory function is then categorized into one of the following: normosmia, mild microsmia, moderate microsmia, severe microsmia, anosmia, and probable malingering.

2. Sniffin' sticks test

In this test, the nasal chemosensory performance is tested with pen-like odour dispensing devices. They comprise of three tests of olfactory function, such as odour threshold, odour discrimination, and odour identification. These three subgroups yield a global olfactory score and the results are calibrated according to gender and age specific normal ranges. The odorants were impregnated in felt-tip pens. Odour thresholds were tested with n-butanol by a single-staircase procedure with sixteen dilutions. Three pens were given randomly among which two had the solvent and the third with the odorant, and the patients were asked to identify the pen with the odorant. Batches of three pens were presented at 20 seconds interval. The patients’ scores ranged from 1 to 16. Then odour discrimination was assessed by presenting triplets of odour

containing pens containing the same and a different odorant. Subjects had to determine which of three pens smelled differently. The scores ranged from 1 to 16.

Thirdly, odour identification was tested using a multiple-choice task identification of 16 different odours. The scores ranged from 1 to 16. The sum of the three tests odour threshold, discrimination, and identification measures are together combined as 'TDI Score' (16).

3***. CCCRC (Connecticut Chemosensory Clinical Research Center) olfactory test***

The test is portable and inexpensive and consists of two components:

1. *Butanol threshold test:* The threshold of subjects for butanol is assessed by means of squeeze-bottles using the method of ascending limits. Patients are presented with two bottles for each trial, one with water and the other with dilute concentrations of butanol. The n-butyl alcohol is an ideal odorant because it is readily soluble in water, less toxic and has neutral odour quality. The strongest butanol concentration (bottle 0) is 4 % butanol in deionized water. Each subsequent dilution (bottles 1–9) is a 1:3 dilution with deionized water.

The solutions are given for sniffing in a 250 ml squeezable polyethylene bottle. Each bottle contains 60ml of the test solution with a popup snout that fits into the nostril. To test the sample, the patient has to place the snout into the nostril and then the bottle is squeezed, while being sniffed.

A testing batch consists of an empty bottle to learn the sniffing technique, two bottles in every concentration and six bottles of deionized water as controls. The patient

is presented with the test concentration and blank deionized water and had to identify which smelt stronger. If the answer is wrong, then a higher concentration is presented. Four correct answers in a row are taken and test is stopped. The total scores ranged from 0 to 9, but all scores 7 and higher were scored as 7 per the CCCRC test.

2. Odour identification test: Eight odours namely cinnamon, asafoetida, coffee, tea, pepper, clove oil, baby powder and vicks are given in opaque jars and the patients are asked to identify the odours one nostril at a time. A list of items which containing the above substances and also another 8 distractors are given in a paper before starting the test. Apart from the correct and wrong response, 'no response' and 'don't know' are also included in the results. Two trials are given for each nostril for every odour. The scores from both the nostrils are averaged to arrive at a final score. Vicks indicates intact trigeminal nerve function; hence it will not be included in the scoring process.

Based on the results of the 2 components of CCCRC test, a composite score is calculated and diagnosis of anosmia, hyposmia and normosmia are made.

Composite score:

0 to 1.75 - anosmia

2 to 3.75 - severe hyposmia

4 to 4.75 - moderate hyposmia

5 to 5.75 - mild hyposmia

6 to 7 - normosmia

As the CCCRC test is easy to perform and can be administered within a few minutes, it is a preferred test for assessment of olfaction (17).

4. *Electro-olfactogram (EOG)*

Electro-olfactograms are electrical potentials of the olfactory epithelium which are produced in response to olfactory stimulation. It displays the total sum of generator potentials of the olfactory neuroepithelium. The EOGs display the role of the central nervous system in olfactory desensitization, topographical distribution of olfactory receptors and also the specific expression of olfactory receptors in response to the respective odorants. Thus, it sums up the electrical potentials of the olfactory receptor neurons.

The above-mentioned tests were psychophysical while this is an objective test. The EOG was first demonstrated and termed by Ottoson in 1959. It is carried out by endoscopically placing an electrode on the olfactory epithelium. The olfactory stimuli is presented in a high concentration for less than 20 milliseconds, but without mechanically inducing sensations in the olfactory epithelium and the response was recorded (18).

OLFACTORY DYSFUNCTION

The ability of human beings to thoroughly know our surroundings is dependent on the sense of smell. The qualities of a well-functioning olfactory system ranges from the ability to identify dangerous situations like fires to remembering a fond memory

triggered by different odours. It also gives the pleasure of eating and helps to avoid spoiled food.

Olfaction also plays a vital role in everyday life by being the most important sensation. There are various effects smell and therefore, the loss of smell can diversely affect the patients. It ranges from markedly reducing the quality of life to being life threatening because the patients cannot identify hazardous situations.

In a study conducted among 22 anosmia patients in Austria, it was found that these patients have reduced quality of life, low body related self-esteem and depressive symptoms. The consequences of loss of smell not only affect the patients, but also bring a burden for the family and the health system (12).

The disorders of smell are frequently identified several months after the onset of the symptoms. Some of the surveys conducted in the United States have described the prevalence of olfactory dysfunction being 1 - 4%, but more recent statistics point out that are more than 20% of them general population are affected by the inability to perceive smell (19). Hence it is essential to identify and help in making guidelines for prompt diagnosis and management of olfactory dysfunction.

The olfactory receptors are activated by the central nervous system for the perception of the sense of smell. Normosmia is defined as normal perception of smell. Olfactory dysfunction can be a result of reduced intensity of the stimulus. Olfactory deficits can also be due to distorted perception of stimulus (20). They can be manifested as the following:

DISORDERS OF OLFACTION

Anosmia **-** complete inability of sense the smell

Partial anosmia **-** inability to perceive limited only to certain odorants

Hyposmia **-** reduced perception of smell

Parosmia **-** perception of an unpleasant odour following presentation of the stimulus

Heterosmia **-** where all the odorants smell similar

Phantosmia **-** perception of smell without the presence of a stimulus

Hyperosmia **-** abnormal increased sensitivity to perceive odours

Dysosmia **-** perception of distorted olfactory sensation

Olfactory agnosia **-** inability to recognize odours despite a normal functioning olfactory system

Prebyosmia **-** age related decline in the perception of smell

Osmophobia **-** fear, dislike or aversion in perceiving odours

TYPES OF OLFACTORY DYSFUNCTION

It is divided into three groups based on the etiology: conductive, sensorineural and central

CONDUCTIVE TYPE

Conductive type of olfactory dysfunction is due to the prevention of odorants from reaching the olfactory epithelium in the roof of nose and the olfactory receptors because of certain anatomical barriers. This is seen in obstructive nasal diseases like

chronic rhinosinusitis, allergic rhinitis, nasal polyposis and nasal masses. These can hinder the nasal airflow towards the olfactory cleft.

Olfactory dysfunction is seen in 61 to 83% of chronic rhinosinusitis patients and they are more prone to develop loss of smell if the age is above 65 years, associated with nasal polyposis and with a history of smoking. The pathology responsible for the higher incidence of olfactory dysfunction in chronic rhino sinusitis patients is attributed to the combined effect of nasal obstruction by the polyps or edematous mucosa and injury caused by inflammation. The olfactory impairment in chronic rhinosinusitis patients is attributed to the level of mucosal eosinophilia by Soler, et al. (21).

SENSORINEURAL TYPE

Sensorineural type of olfactory dysfunction is due to reduced reception or processing of the stimulus by one or more of the components in the olfactory pathway which includes olfactory receptors, olfactory neurons or the centers of olfaction in the central nervous system. Examples include viral infection, age related, drug induced or congenital.

Other causes of anosmia include head trauma, in which 20 - 30% patients develop some amount of olfactory impairment, while 5% develop complete olfactory loss. The mechanism involves injury induced hematoma, contusions and most importantly shearing of the olfactory nerves and tracts. The coup or the contra coup movement of the brain in relation to the skull causes extensive shearing of the olfactory nerve filaments at the level of the cribriform plate. The return of sense of smell following head trauma in majority of patients is incomplete which is due to the

incomplete regeneration of the olfactory neuroepithelium and the tracts traveling to the olfactory bulb through the cribriform plate. In turn, the damaged olfactory epithelium is disrupted and replaced by respiratory epithelium, where the number of ciliated olfactory receptor cells are reduced in number (22).

There is presence of recent history of upper respiratory tract infections in 20 - 30% patients with olfactory dysfunction. The frequent causative agents for upper respiratory infections include influenza virus, rhinovirus, parainfluenza virus and respiratory syncytial virus. The relationship between the viral infection and loss of smell is not well understood yet.

There are a few postulated mechanisms including scarring of the olfactory epithelium following viral infections. There is also evidence of replacement of the olfactory epithelium with the respiratory type epithelium. In such conditions, the receptors were found abnormal. The dendrites in those receptors did not reach the surface of the epithelium and also lacked sensory cilia, which is the site of odorant transduction. These patients also had reduced number of olfactory receptors (23).

In a study conducted by Yamagishi in Japan on 13 patients, the author has described three patterns of olfactory neuroepithelium in patients who had olfactory dysfunction following viral respiratory infections. In the first pattern, the basic cellular structure of the epithelium was preserved but had a smaller number of receptor cells. The second pattern showed thinner olfactory epithelium which consisted only of supporting cells and basal cells. While the third pattern, which is also most severe among the three showed that the neuroepithelium was replaced by metaplastic squamous epithelium (24).

Olfactory dysfunction is also noted in increasing age among the human population. The incidence is around 62 - 80% in people more than 80 years of age. The pathology behind the smell loss is multifactorial resulting in a decrease in odour identification, quality discrimination and threshold detection. The following possible etiologies have been proposed as the cause for age related loss of smell. They are high expression of a proapoptotic gene in the olfactory epithelium, age related narrowing of the foramina in the cribriform plate and degeneration of olfactory epithelium with increasing age (20).

Isolated congenital anosmia is seen in 1 in 10000 people, and it is characterized by loss of sense of smell since birth in otherwise healthy individuals. It requires detailed history, examination with electrophysiological and radiological investigations to arrive at a diagnosis. Radiological workup has shown hypoplastic or aplastic olfactory bulbs with shallow olfactory sulcus in otherwise healthy people. These patients are unaware of the olfactory loss in childhood while the parents are suspicious that they are not aware of the spoiled food or toxic environmental chemical odours (25).

Other causes of sensorineural type of olfactory dysfunction include Kallmann syndrome, which is characterized by hypogonadotropic hypogonadism. Medications like angiotensin converting enzyme inhibitors frequently cause olfactory dysfunction. Other medications that are reported to cause smell loss are diuretics, calcium channel blockers and statins. Environmental air pollutants and industrial dusts like ashes, benzene, chromium, lead, nickel, silicone dioxide and many others also cause loss of smell.

There are multiple types of chemical agents which can cause loss of smell like cadmium, formaldehyde, benzene, solvents and nickel dust. It is found that in approximately 5% of people that present with olfactory loss to a hospital there is direct relationship with exposure to a toxic compound.

Tobacco smokers have olfactory loss and is directly proportional to the cumulative smoking dose and the duration of smoking. Also, cessation of smoking promises improvement in the olfactory ability over time.

CENTRAL TYPE

Tumours in the region of olfactory bulbs or tracts can cause significant olfactory dysfunction. They are olfactory groove meningioma, frontal lobe glioma and suprasellar ridge meningioma seen arising from the dura of the cribriform plate. The vision is frequently affected along with the smell as the olfactory nerve is in close proximity to the medial wall and roof of the orbit, optic nerve and tracts.

Foster- Kennedy syndrome results from mass lesions around the olfactory region and is characterized by ipsilateral anosmia, ipsilateral optic atrophy and contralateral papilledema due to increased intracranial pressure. Tumours in the olfactory processing area like the medial temporal lobe mass has the potential to present with loss of smell. The hallmark of olfactory loss seen in association with tumours is presence of headache as an accompanying symptom.

Granulomatous diseases like syphilis, systemic lupus erythematosus, granulomatosis with polyangiitis, sarcoidosis also cause anosmia. Lymphoma infiltrating the olfactory area in the roof of nose also present with loss of smell.

The following are some examples intranasal neoplasms causing olfactory dysfunction: esthesioneuroblastoma, neurofibroma, schwannoma, paranasal and nasopharyngeal tumours with extension. Neoplasms involving the distant organs like lung, ovary, testes, larynx and gastrointestinal tract can also present with anosmia.

Olfactory dysfunction is seen frequently in a multitude of neurological diseases. Neurodegenerative disorders like Alzheimer's disease and idiopathic Parkinson's disease also present with loss of smell, frequently the first clinical presentation. In Parkinson's disease, the smell loss is bilateral and it presents early before the classical neurological signs and symptoms. The severity of the loss of smell is not linked to the disease stage, duration of the disease, intake of antiparkinson drugs or the severity of the symptoms.

Graves and colleagues have found that in Alzheimer disease the olfactory dysfunction is in association with one or more APOE-e4 alleles. This association is predicted to carry a very high risk of cognitive decline compared to the patients who did not have olfactory dysfunction (26).

Multiple sclerosis is also a cause of central type of olfactory dysfunction. The severity of the loss of smell in multiple sclerosis is directly proportional to the number of demyelinating lesions in the brain closely related to the regions of olfactory processing like inferior middle temporal lobe and periorbital frontal cortex. The loss of smell fluctuates according to the number of plaques in the nervous system.

There is significant amount of deficiency in the odour identification capacity and olfactory threshold sensitivity in patients with schizophrenia. There is an inverse relationship between the disease progression and the olfaction testing scores, which

predicts possible progressive neurodegenerative changes involving the olfactory pathways in schizophrenia. Hence it can be used as a marker of progression of disease.

Olfactory auras or hallucinations are seen rarely in association with headache and seizures. They occur as sudden unpleasant smell sensation. Anosmia is also seen in temporal lobe epilepsy, which houses structures like amygdala and hippocampus responsible for the processing of olfaction.

In a study done among 3100 diabetic patients over the age of 40 in the United States, it was found that a significant number of them had severe hyposmia or anosmia. There is increased incidence of cortical thinning in the orbitofrontal cortex as a result of hyperglycemia and type 2 diabetes has been associated with cortical and subcortical atrophy. Phantosmia is linked with loss of grey matter volume at the orbitofrontal cortex, hence it is thought that diabetes can exaggerate the risk of olfactory dysfunction by affecting the same region in the brain (27).

Another study done in Greece among 154 diabetics, showed reduction in odour threshold and odour identification. This was seen more in association with diabetic peripheral neuropathy and retinopathy which are due to microangiopathy. Hence it was postulated that microangiopathy is the cause for olfactory dysfunction among diabetic patients (28).

The relationship between smell dysfunction and hypertension is thought to be due to the alteration of the chemosensory function. The antihypertensives play a major role in altering the chemosensory function, which in turn causes smell and taste disturbance. The exact mechanism by which this results is yet to be known (29).

The relationship between allergic rhinitis and the occurrence of olfactory loss has been studied in a group of 90 patients in Philadelphia. It was found that there is increased incidence of damage to olfactory epithelium due to frequent respiratory infection in allergic rhinitis patients. This results in olfactory dysfunction more than the patients with other nasal diseases like chronic rhino sinusitis and nasal polyps (30).

There is increased occurrence of smell loss among chronic rhinosinusitis patients which accounts for the most common cause of smell loss. There are many mechanisms that can lead to reduced olfactory ability like changes in mucus composition, effects of inflammatory mediators at the olfactory receptor level and altered air flow to the olfactory region (31).

TREATMENT OF OLFACTORY DYSFUNCTION

There are many successful treatments available for the anosmia that is caused primarily by the airflow obstruction through the nose to the olfactory neuroepithelium. The most common causes for conducive anosmia include sinonasal disease, infections and nasal masses. Sensorineural type of anosmia is not easy to treat and the prognosis is poor in patients having a chronic course following upper respiratory infections and trauma to head.

CORTICOSTEROIDS

Patients with conductive smell loss often respond well with oral corticosteroid therapy, whereas sensorineural loss is refractory. The chronic rhino sinusitis related smell loss improves by corticosteroid related modulation of sodium potassium

adenosine triphosphate which is present in the olfactory receptor neurons. In cases of anosmia related to upper respiratory tract infections, a combination of topical and systemic corticosteroids is proven to improve the olfaction (20).

The reason behind the failure of improvement in smell loss by topical nasal steroids is that they are unsuccessful in reaching the olfactory region which is situated in the roof of the nasal cavity. There has been seen increased efficacy if steroids are delivered in formulations like drops or sprays in the head down position.

In a study conducted in Germany, among 92 patients with olfactory dysfunction where 55 of them were prescribed oral prednisolone course while the rest 37 used mometasone steroid spray. The conclusions were that local administration of steroid sprays improve only the odour identification but did not effect the olfactory function. But in contrast, systemic steroids significantly improved the olfactory function in all categories of causes even in post upper respiratory tract infections and idiopathic olfactory dysfunction. The other conclusion was that the response to oral corticosteroids were unaffected by the duration of disease, age and gender of patients, or the association of parosmia (32).

OLFACTORY TRAINING

The olfactory training is a therapeutic approach which is attractive due to its simplicity, low cost and lack of potential side effects. It is done by regular structured exposure to different odours for a period of 12 weeks. Four ml of four odours namely lemon, rose, cloves and eucalyptus are soaked in cotton pads and placed in four amber coloured glass bottles of 60ml capacity and given to the patients.

Patients are instructed to open each glass bottle, hold it under their nose, and breathe slowly and deeply for 15 seconds. They are advised to perform this training method twice a day, preferably after a meal every day for 3 months. All the glass bottles are named with the respective odorant. Olfactory testing is done before and after the olfactory training.

In a study done in 56 patients, 40 patients performed olfactory training and 16 patients did not. Compared to the baseline, patients who performed olfactory training experienced an increase in their olfactory function while the olfactory function remained unchanged in patients who did not perform training. It appears to increase the olfactory function in approximately 30% as compared to the patients with no olfactory training. Olfactory training is well tolerated and easy for patients to perform on their own and can be offered to any patient who complains of olfactory loss, regardless of the cause (33).

ALPHA LIPOIC ACID

Alpha-lipoic acid is a fatty acid and it crosses the blood brain barrier. It is metabolized to dihydrolipoic acid which is the active metabolite as soon as it is taken up by the cells, and then released into the extracellular space. One of the actions of the active metabolite is to reduce the levels of superoxide and peroxide radicals. Dihydrolipoic acid restores thioredoxin and vitamin C helps regenerate vitamin E by raising the intracellular glutathione concentration.

Therefore, both alpha lipoic acid and dihydrolipoic acid acts towards repairing the oxidative lesions in the body. It has been approved for the treatment of diabetic

neuropathy and has been found safe. The possible mechanism of action is release of nerve growth factor, substance P and neuropeptide Y which increases the motor nerve conduction and microcirculation which results in the improvement of the smell.

Oral alpha lipoic acid at a dose of 600mg/day taken for 3 to 11 months showed significant improvement in the olfactory function. In a study done in 23 patients, 19 of them were hyposmic and 4 had anosmia. They took 600mg/ day of oral alpha lipoic acid for an average period of 4.5 months. Six patients (26%) showed moderate and eight patients (35%) showed remarkable increase in olfactory function (34).

ZINC

Zinc is a trace metal which exerts enzyme activity involved in cell proliferation. Hence, it has been proposed that zinc has an important role in the olfaction and taste as it helps in regeneration of the sensory cells. Patients who undergo chemotherapy often develop loss of smell and its due to inhibition of various growth factors that participate in the sensory functions of olfactory neuroepithelium. Zinc has been used in these patients as it acts as one such growth factor and improves the olfaction.

In a study done in 95 patients with post traumatic olfactory loss, participants were categorized into three groups. One group received Zinc only, second had a combination of zinc sulphate along with the conventional treatment (vitamin B with topical corticosteroids) and the last group received only the conventional treatment. It showed significant improvement in olfaction rates in the zinc sulphate group compared to those who took the conventional therapy (35).

VITAMIN A

Vitamin A is involved in the functional regeneration of smell due to the role of retinol in the olfactory receptor neurons. Studies have shown evidence of cell death among olfactory receptor neurons with defective retinoid acid receptors. Hence, retinoic acid is formulated to stimulate immune response and help in the fast recovery following damage in the olfaction system. The reason is the capacity of retinoic acid to regenerate by modulation of gene expression (36).

In a study conducted by Duncan and Briggs in 56 patients of olfactory loss, 50 of them showed improvement following treatment with Vitamin A. The oral formulation was found helpful in the maintenance while injectable vitamin A yielded good improvement in the olfaction. The vitamin A plays a vital role in the olfaction and the site of action is the vesicle and process of the olfactory cells. It also returns the normal adaptation rate of the olfactory mucosa (37).

THEOPHYLLINE

Theophylline is a non-specific phosphodiesterase inhibitor and acts by increasing the intracellular cAMP levels in the neurons. In the patients with olfactory dysfunction, it was seen that the levels of cAMP and cGMP in the saliva and nasal mucous were below normal range. These cyclic nucleotides function as growth factors for various neural tissues, including the olfactory epithelium.

Hence a study was conducted with treating two groups of anosmia patients, one with theophylline and the other group without it. The phosphodiesterase inhibitor was given to elevate the levels of cyclic nucleotides in the nasal mucosa and saliva. It was

found that theophylline corrected hyposmia in many of the patients, which was seen by psychosocial measurements of olfaction (38).

MINOCYCLINE

Minocycline belongs to the tetracycline group of drugs which are primarily used in the treatment of infections of upper respiratory and urogenital system. Apart from the antibiotic and anti-inflammatory property, it also has effects on the apoptotic pathway in the neurons. It has been used in various other conditions like Huntington's disease, Parkinson's disease and cerebral ischemia due to the neuroprotective potential.

One of the proposed causes for olfactory loss is the reduction in the number of mature and functional olfactory neurons in the olfactory neuroepithelium. This reduction is due to the possible apoptosis that occurs more readily due to their exposed location. Thus, minocycline is thought to inhibit the apoptosis occurring in the olfactory neurons and thereby increase the total number of the olfactory receptor neurons (39).

ANTIHISTAMINES

There is a direct relationship between the severity of persistent allergic rhinitis and the loss of smell. A randomized control trial has shown that antihistamines greatly improve the visual analogue scores in patients with olfactory dysfunction (40).

OMALIZUMAB

Omalizumab is an anti-immunoglobulin directed against the IgE antibody. In patients with olfactory loss associated with chronic rhinosinusitis and bronchial asthma,

this drug has shown good results. In a study, subcutaneously administered omalizumab for a period of 16 weeks has shown increase in the olfactory awareness scores compared to placebo in olfactory loss patients (41).

ROLE OF SURGERY

Olfactory dysfunction secondary to sinonasal polyposis benefit good improvement in olfaction following endoscopic sinus surgery.

AIMS AND OBJECTIVES

AIMS AND OBJECTIVES

AIM

The aim of this study is to analyze the variations in *n*-butanol threshold and odor identification scores of the CCCRC test (Connecticut Chemosensory Clinical Research Centre) in patients with various olfactory disorders.

OBJECTIVES

Primary objective

a**.** To determine *n*-butanol olfactory threshold in different olfactory disorders

b. To determine smell identification score in different olfactory disorders

Secondary objective:

To analyze the relative change in olfactory threshold and odor identification scores in different olfactory disorders

METHODOLOGY

METHODOLOGY

STUDY DESIGN

This is a retrospective observational cohort study done in a tertiary hospital.

STUDY POPULATION

Patients for this study were recruited retrospectively from institutional database. All patients who were diagnosed with olfactory dysfunction, between January 2018 and March 2020, based on CCCRC olfaction testing were selected retrospectively after applying the inclusion and exclusion criteria.

INCLUSION CRITERIA

Patients above 16 years of age who underwent olfaction testing from January 2018 to March 2020 and were found to have olfactory dysfunction

EXCLUSION CRITERIA

Patients with normal olfaction and children

STUDY PERIOD

January 2018 to March 2020

PATIENT RECRUITMENT

All patients fulfilling the inclusion and exclusion criteria were recruited and then the details of each patient including demography, presenting symptoms, duration of symptoms, clinical examination and olfaction testing details were recorded. At the end

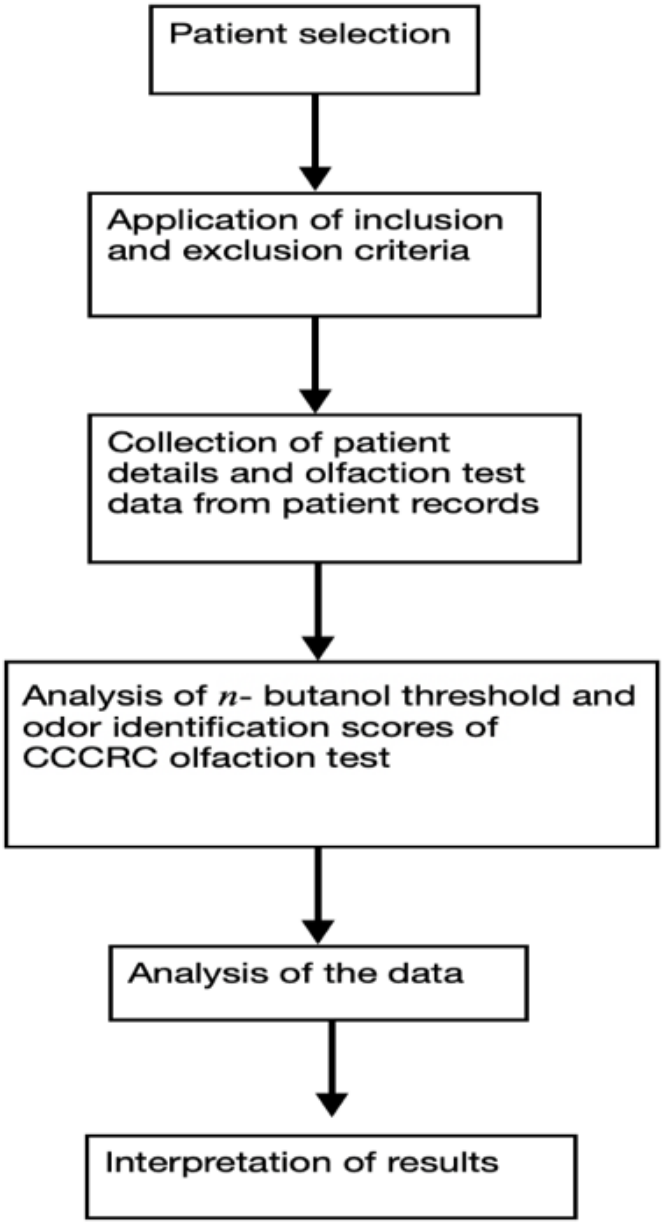
of the study, the CCCRC test *n*-butanol threshold scores and the odour identification scores were analysed for variations in each of the olfactory disorder and in various systemic diseases.

CCCRC test is a test in which, the threshold of subjects for butanol is assessed by means of squeeze-bottles using the method of ascending limits. Subjects will be instructed to occlude one nostril. The participant is presented with a bottle with the test concentration and a blank bottle with water and has to decide which smelled stronger. If incorrect, the participant receives another blank paired with the next higher concentration. Errors trigger increments in concentration whereas correct choices led to another presentation of the same concentration and a blank. Four correct choices in a row led to cessation of testing and the concentration at which this occurred is taken as the olfactory threshold. Now testing is switched to the other nostril. The scores for both nostrils will be averaged to arrive at the final score. The strongest butanol concentration (bottle 0) is 4 % butanol in deionized water. Each subsequent dilution (bottles 1–9) is a 1:3 dilution with deionized water. Possible scores ranged from 0 to 9, but all scores 7 and higher were scored as 7 per the CCCRC test.

Odour identification is performed by means of eight bottles containing different odorants with a multiple choice from a list of 16 items identical for all odorants.

The analysis on the change in parameters with regard to patient related factors like gender, age and sex. The analysis based on pre-existing systemic conditions like diabetes, hypertension, dyslipidemia and hypothyroidism are also done.

DETAILED DIAGRAMMATIC ALGORITHM OF THE STUDY



RESULTS

RESULTS

A total of 245 patients were recruited for this study. All patients underwent olfaction testing by CCCRC (Connecticut Chemosensory Clinical Research Centre) olfaction test. As each nasal cavity was assessed separately, the total sample size was taken as 490.

Types of olfactory dysfunction:

Based on the results of the two components of the CCCRC test, the composite score was calculated. A diagnosis of anosmia, severe hyposmia, moderate hyposmia, mild hyposmia or normosmia was made as follows.

Composite score:

0 to 1.75 - Anosmia

2 to 3.75 - Severe hyposmia

4 to 4.75 - Moderate hyposmia

5 to 5.75 - Mild hyposmia

6 to 7 - Normosmia

Nasal cavities with normosmia were excluded from the analysis. In the cohort maximum number of patients had anosmia followed by severe hyposmia. The incidence of different grades of olfactory loss is shown in Table 1.

Table 1: Incidence of olfactory dysfunction

Types	Anosmia	Severe hyposmia	Moderate hyposmia	Mild hyposmia
Number	231	102	87	63
Percentage	47.14%	20.82%	17.76%	12.86%

(Note: Normosmia is not shown here)

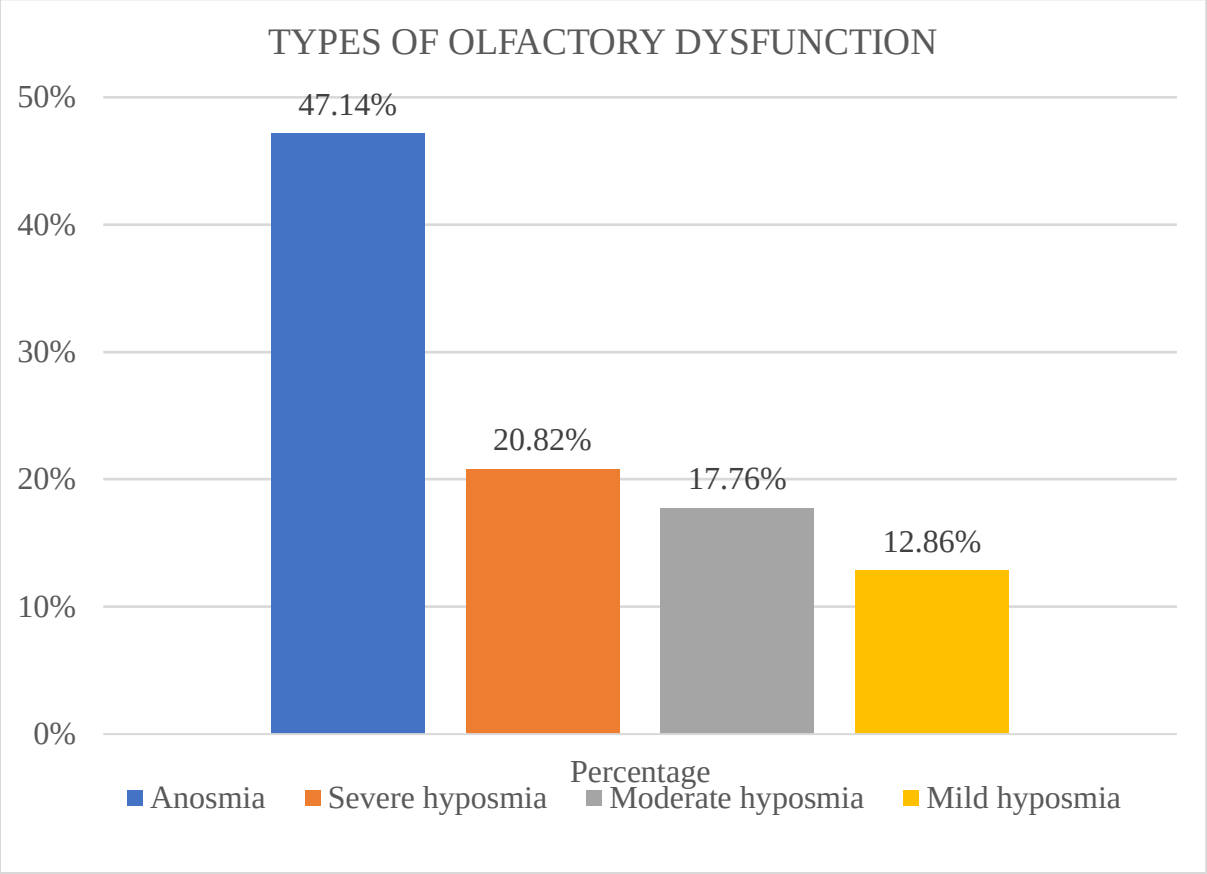


Figure 12 - Incidence of olfactory dysfunction

Demographics:

Age

Age ranged from 18 to 72 years with a mean of 41 years in the entire cohort.

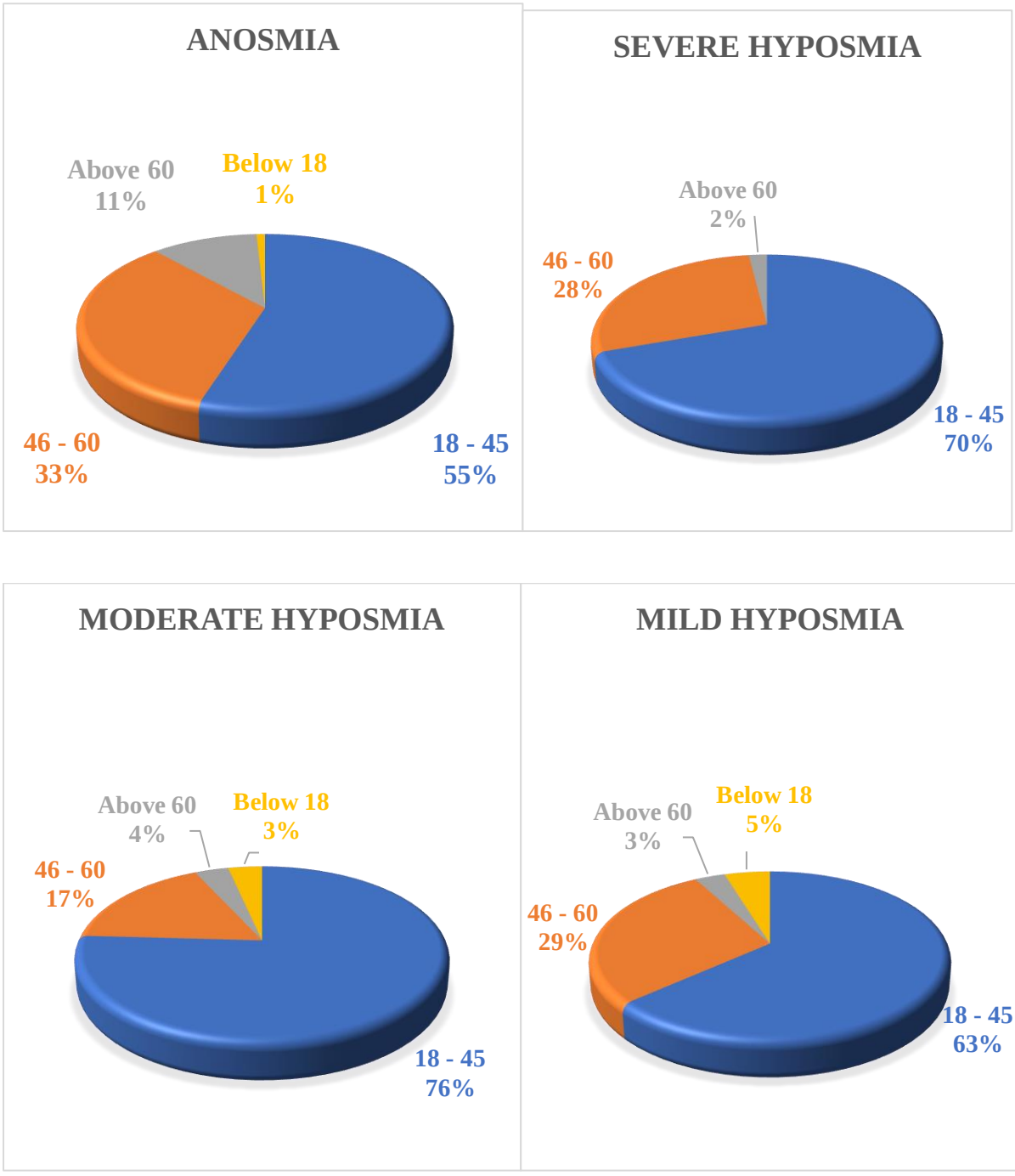


Figure 13 - Age distribution

Majority of patients in all four types of olfactory dysfunction fell in the 18-45 age group.

Gender

The study population consisted of 151 males (61.63%) and 94 females (38.37%). Among the patients, all the four grades of olfactory dysfunction were seen predominantly in the male population.

Table 2: Gender distribution

Sex	Anosmia		Severe hyposmia		Moderate hyposmia		Mild hyposmia	
	No.	%	No.	%	No.	%	No.	%
Male	67	58.26%	35	64.81%	25	69.44%	22	61.11%
Female	48	41.74%	19	35.19%	11	30.56%	14	38.89%

Prevalence of comorbidities

The comorbidities prevalent among the study population were diabetes mellitus, hypertension, hypothyroidism, bronchial asthma and dyslipidemia. The most common comorbidity was found to be systemic hypertension followed by diabetes mellitus.

Table 3: Prevalence of comorbidities

Comorbidities	Diabetes mellitus	Hypertension	Hypothyroidism	Bronchial asthma	Dyslipidemia
Number	52	74	22	22	10
Percentage	10.61%	15.10%	4.49%	4.49%	2.04%

(Note: Percentage shown is out of 490 samples)

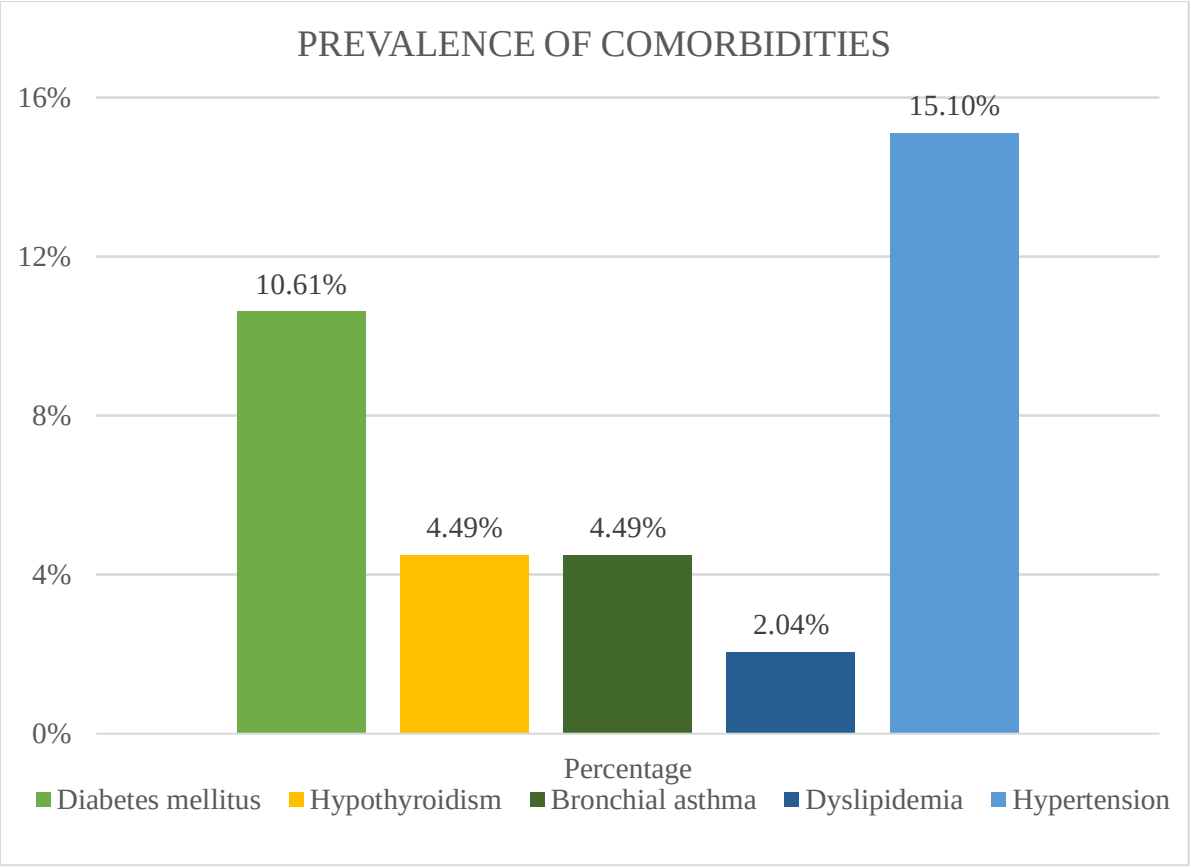


Figure 14 - Prevalence of comorbidities

Olfactory threshold and odour identification:

The two components of CCCRC test are n-butanol threshold and odour identification. The n-butanol threshold has scores from 0 to 6 and odour identification scores range from 0 to 7.

Olfactory threshold in types of olfactory dysfunction:

The distribution of olfactory threshold in all grades of olfactory loss is shown in table 4

Table 4: Olfactory threshold in types of olfactory dysfunction

CCCRC Olfactory threshold	Anosmia N=231	Severe hyposmia N=102	Moderate hyposmia N=87	Mild hyposmia N=63
0	**93.94% (N=217)**	**67.65% (N=69)**	0.00% (N=0)	0.00% (N=0)
1	4.76% (N=11)	14.71% (N=15)	20.69% (N=18)	0.00% (N=0)
2	0.43% (N=1)	13.73% (N=14)	**59.77% (N=52)**	0.00% (N=0)
3	0.87% (N=2)	3.92% (N=4)	10.34% (N=9)	**84.13% (N=53)**
4	0.00% (N=0)	0.00% (N=0)	8.05% (N=7)	9.52% (N=6)
5	0.00% (N=0)	0.00% (N=0)	0.00% (N=0)	6.35% (N=4)
6	0.00% (N=0)	0.00% (N=0)	1.15% (N=1)	0.00% (N=0)

Most of the patients with anosmia and severe hyposmia had a predominant olfactory threshold score of zero. Among patients with moderate hyposmia, a majority had a threshold score of 2 followed by score 1. In the mild hyposmia group, the dominant score was 3.

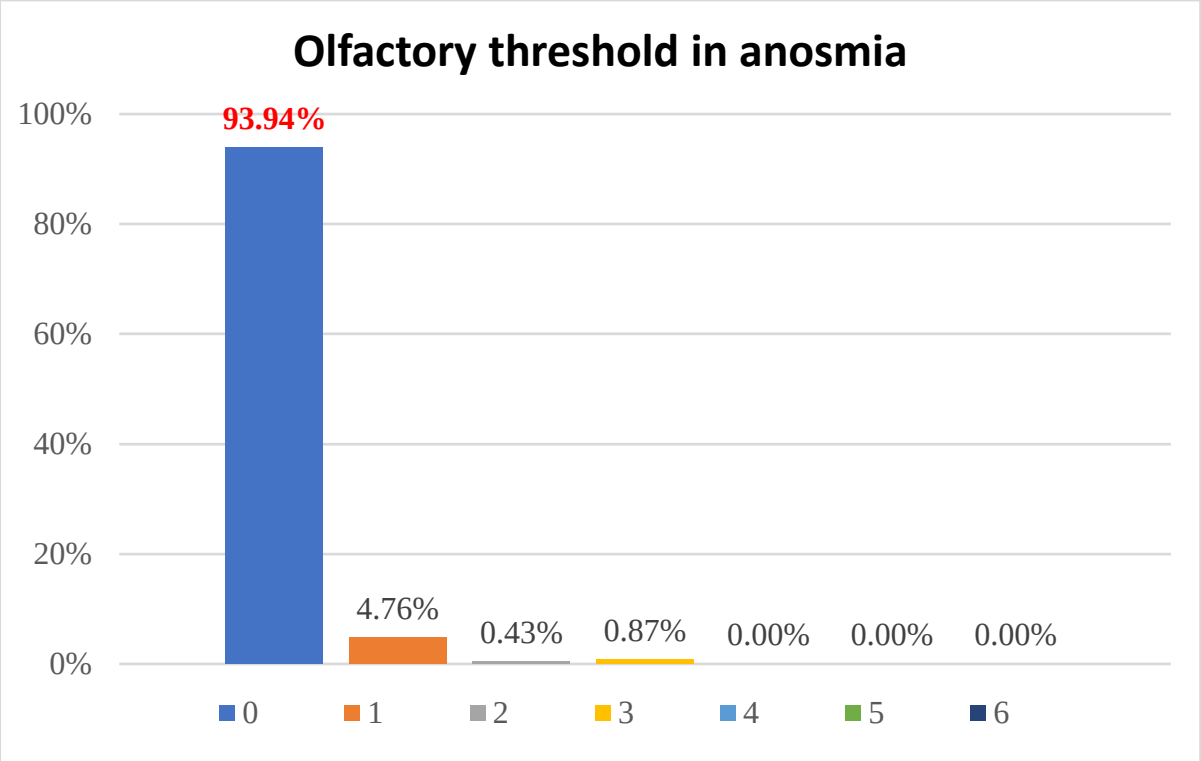


Figure 15 - Olfactory threshold scores in anosmia

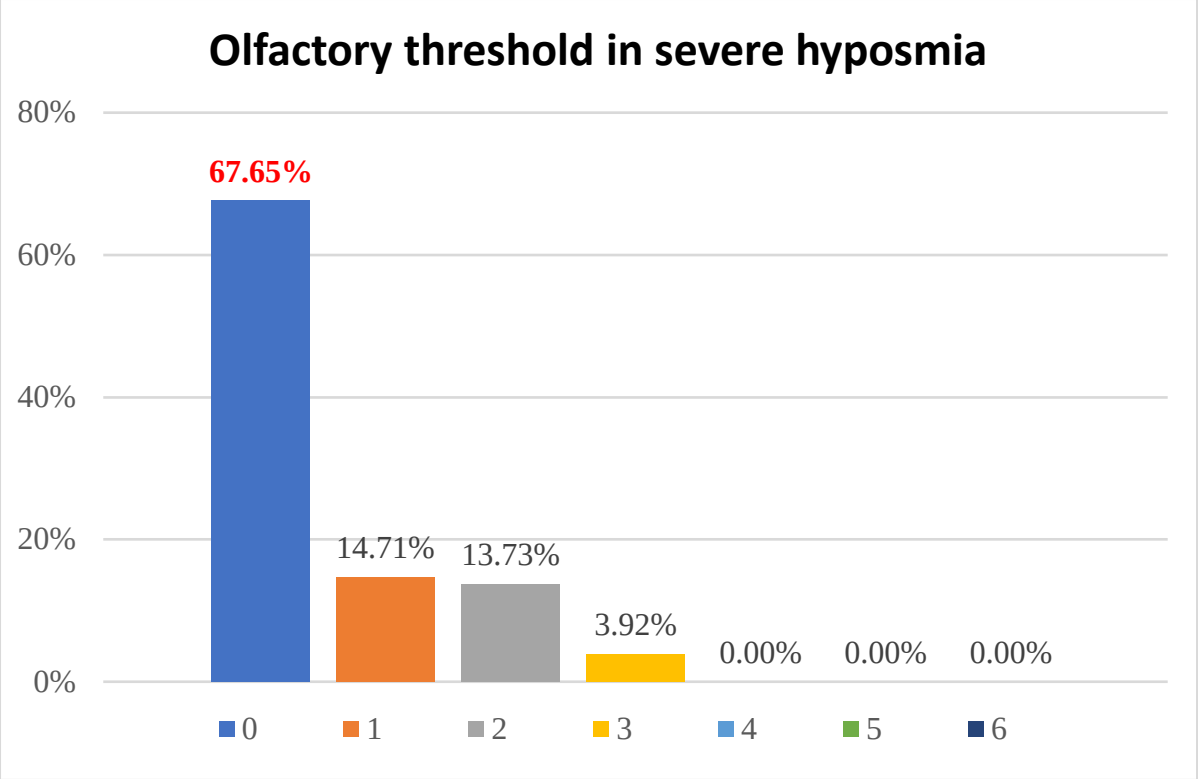


Figure 16 - Olfactory threshold scores in severe hyposmia

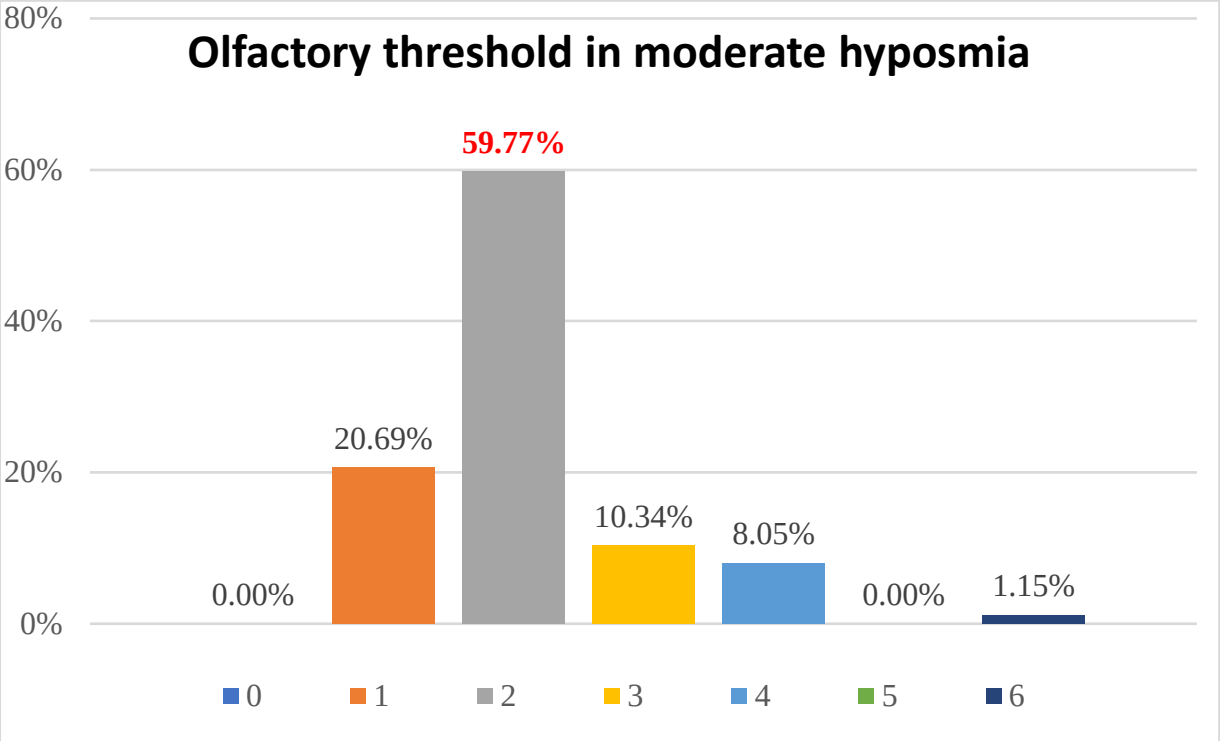


Figure 17 - Olfactory threshold scores in moderate hyposmia

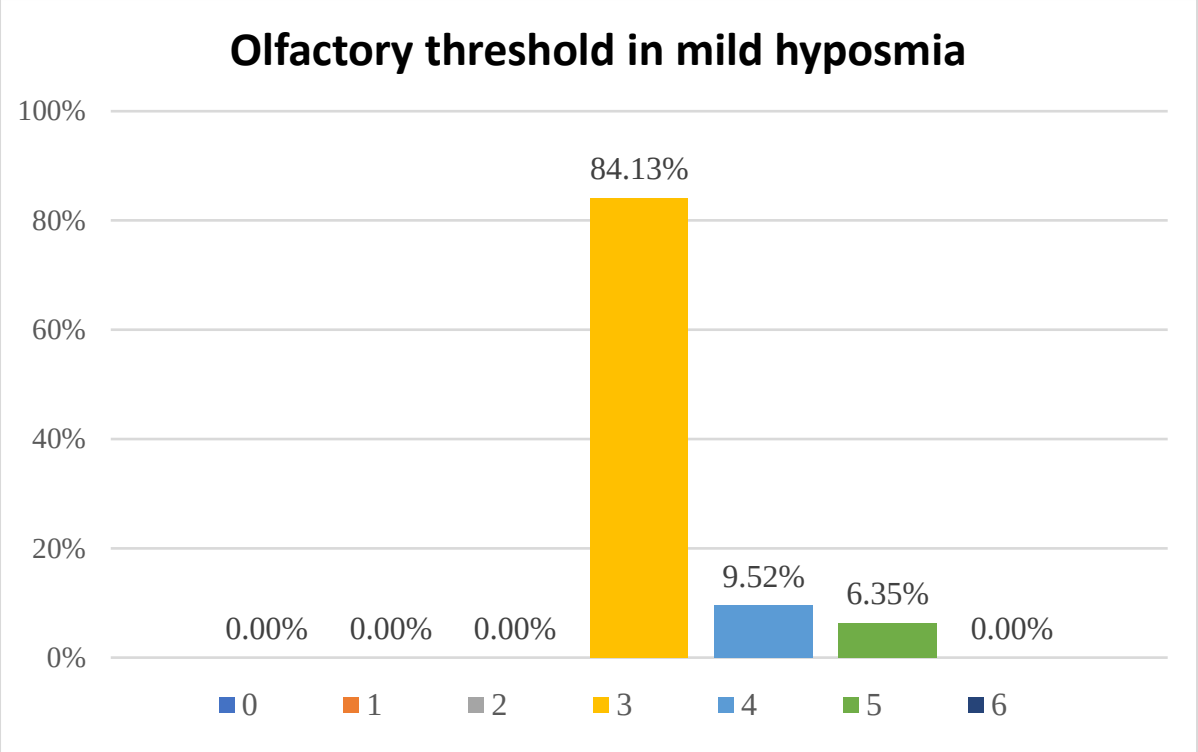


Figure 18 - Olfactory threshold scores in mild hyposmia

Odour identification scores in types of olfactory dysfunction:

The second component of the CCCRC test is odour identification. The scoring was based on the ability of the patient to identify odours (cinnamon, asafoetida, coffee, tea, pepper, clove oil and baby powder). In the study cohort, majority of patients with anosmia had an odour identification score of 0, whereas it was 6 followed by 5 within severe hyposmia group. In the moderate and mild hyposmia population the most common score was 7 (Table 5).

Table 5: Odour identification scores in types of olfactory dysfunction

Odour Identification	Anosmia N=231	Severe hyposmia N=102	Moderate hyposmia N=87	Mild hyposmia N=63
0	**80.95% (N=187)**	0.00% (N=0)	0.00% (N=0)	0.00% (N=0)
1	6.93% (N=16)	0.00% (N=0)	0.00% (N=0)	0.00% (N=0)
2	8.23% (N=19)	0.00% (N=0)	1.15% (N=1)	0.00% (N=0)
3	3.90% (N=9)	7.84% (N=8)	0.00% (N=0)	0.00% (N=0)
4	0.00% (N=0)	17.65% (N=18)	1.15% (N=1)	0.00% (N=0)
5	0.00% (N=0)	**28.43% (N=29)**	10.34% (N=9)	4.76% (N=3)
6	0.00% (N=0)	**28.43% (N=29)**	20.69% (N=18)	6.35% (N=4)
7	0.00% (N=0)	17.65% (N=18)	**66.67% (N=58)**	**88.89% (N=56)**

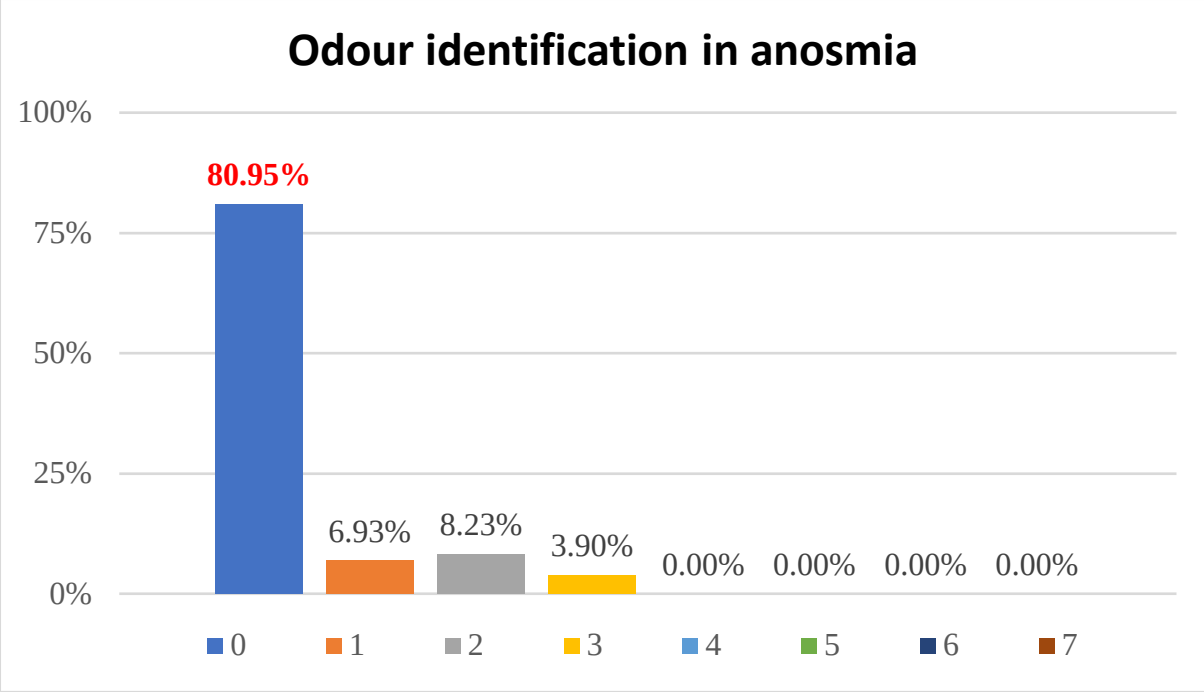


Figure 19 - Odour identification scores in anosmia

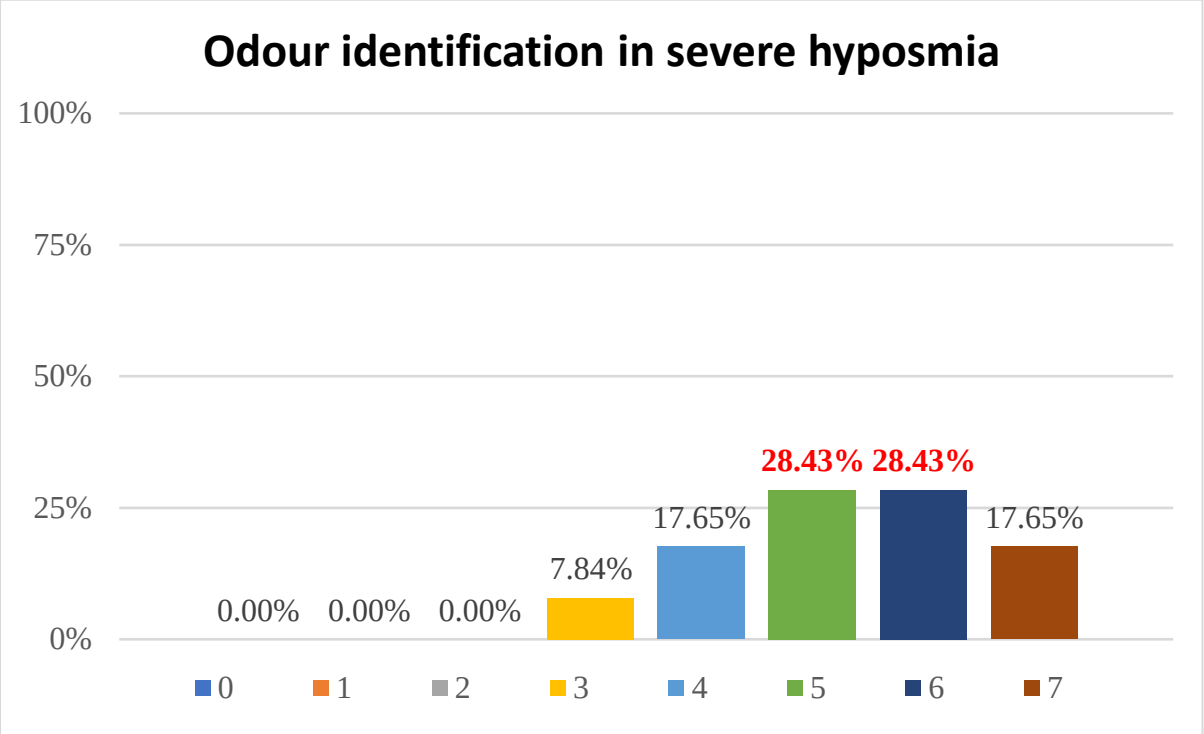


Figure 20 - Odour identification scores in severe hyposmia

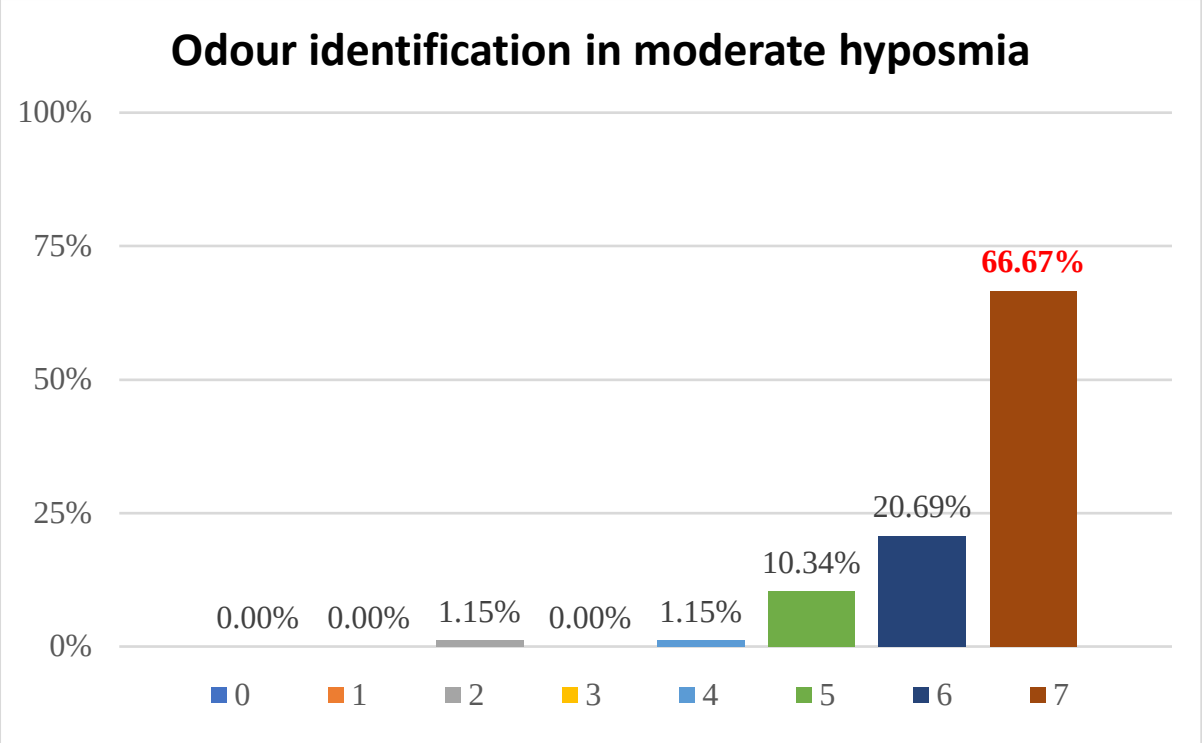


Figure 21 - Odour identification scores in moderate hyposmia

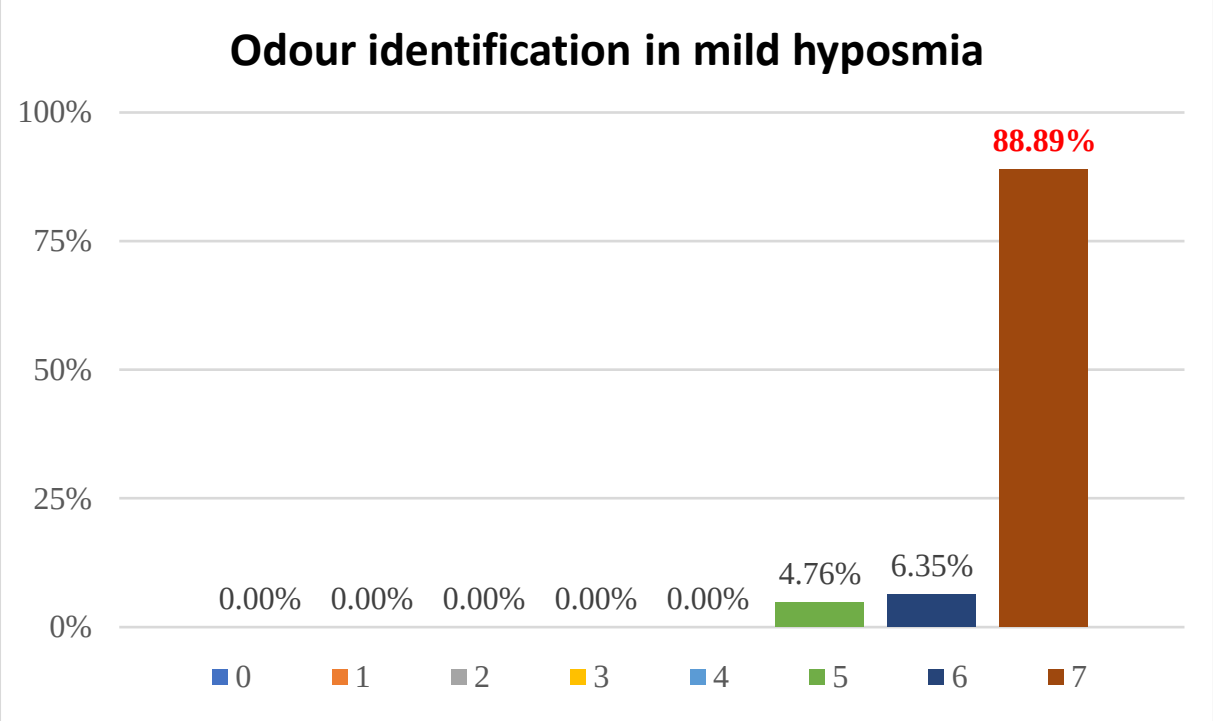


Figure 22 - Odour identification scores in mild hyposmia

Most common odour affected in various types of olfactory dysfunction:

The four different types of olfactory dysfunction namely anosmia, severe hyposmia, moderate hyposmia and mild hyposmia showed specific predilection in the loss of smell. More than 90% of patients with anosmia showed a failure to identify all the seven tested odours. Pepper was the least perceived smell among the patients with anosmia, severe hyposmia and moderate hyposmia. Mild hyposmia group showed less perception of cinnamon among other smells.

Table 6: Odour affected vs olfactory dysfunction

	Cinnamon	Asafoetida	Coffee	Tea	Pepper	Clove oil	Baby powder
Anosmia	91%	90%	95%	97%	**99%**	95%	97%
Severe hyposmia	5%	14%	9%	20%	**65%**	12%	46%
Moderate hyposmia	5%	5%	5%	2%	**17%**	6%	10%
Mild hyposmia	**5%**	3%	0%	3%	3%	2%	0%

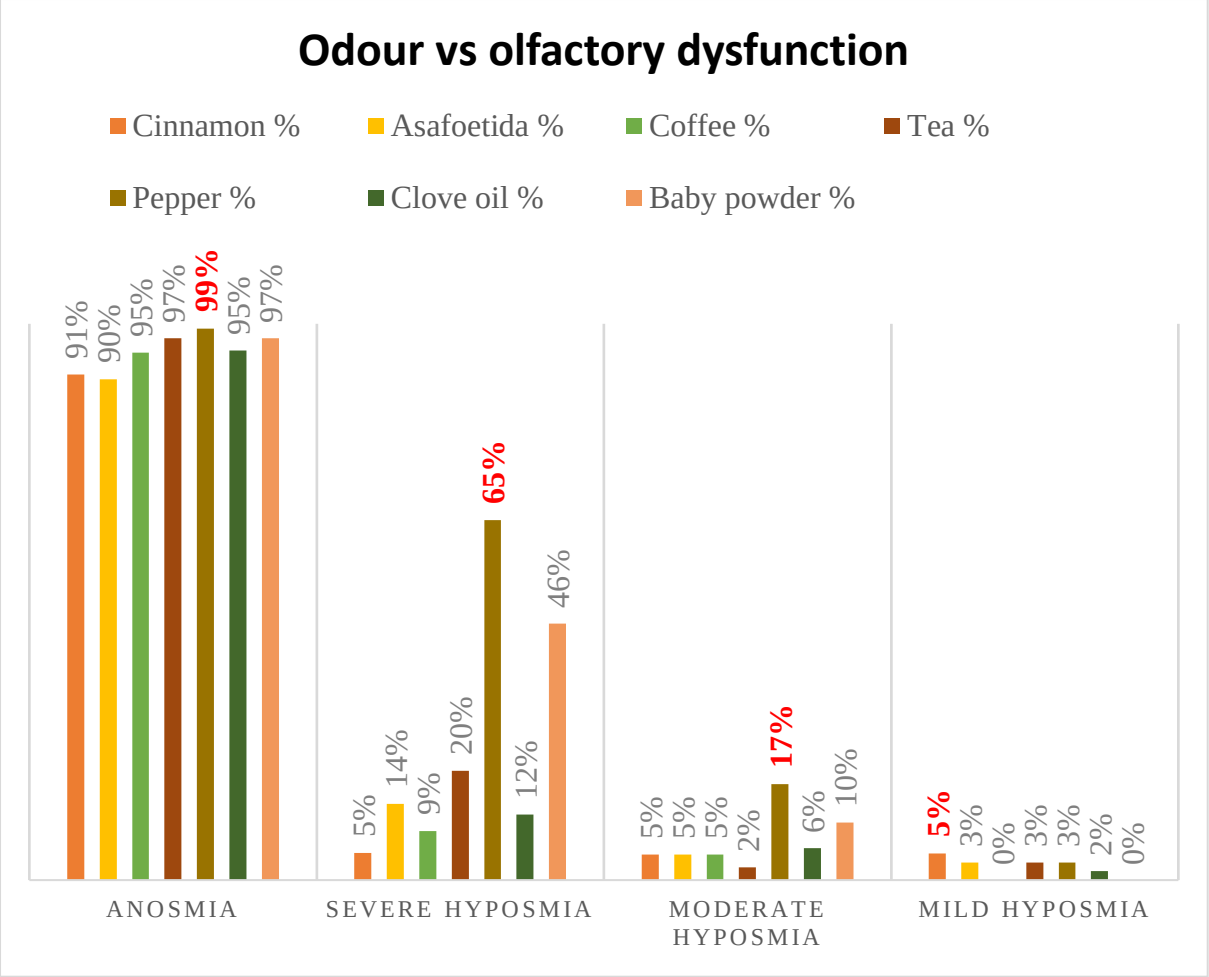


Figure 23 - Odour vs olfactory dysfunction

Co-morbidities

Among the study population the following comorbidities were seen. They were diabetes mellitus, systemic hypertension, hypothyroidism, bronchial asthma and dyslipidemia. Anosmia was seen predominantly among the diabetics, hypertensives and dyslipidemia patients, whereas severe hyposmia was the most common type of olfactory dysfunction seen in hypothyroid patients and moderate hyposmia in asthmatics.

Table 7: Comorbidities vs olfactory dysfunction

Types	Anosmia		Severe hyposmia		Moderate hyposmia		Mild hyposmia	
Comorbidities	No.	%	No.	%	No.	%	No.	%
Diabetes mellitus (N=52)	26	50.00%	9	17.31%	11	21.15%	6	11.54%
Hypertension (N=74)	39	52.70%	9	12.16%	13	17.57%	13	17.57%
Hypothyroidism (N=22)	3	13.64%	11	50.00%	3	13.64%	4	18.18%
Bronchial asthma (N=22)	5	22.73%	4	18.18%	7	31.82%	5	22.73%
Dyslipidemia (N=10)	6	60.00%	0	0.00%	3	30.00%	1	10.00%

Most common odour affected in various comorbidities:

The most frequent smell which was not perceived among each type of comorbidity revealed that the diabetics had difficulty perceiving pepper and baby powder. Among the hypertensives and patients with hypothyroidism, it was pepper. The baby powder was less perceived by the patients who had bronchial asthma and dyslipidemia.

Table 8: Odour identification loss vs comorbidities

	Cinnamon	Asafoetida	Coffee	Tea	Pepper	Clove oil	Baby powder
Diabetes mellitus N=52	42.31% (N=22)	50.00% (N=26)	46.15% (N=24)	55.77% (N=29)	**57.69% (N=30)**	48.08% (N=25)	**57.69% (N=30)**
Hypertension N=74	51.35% (N=38)	56.76% (N=42)	51.35% (N=38)	59.46% (N=44)	**64.86% (N=48)**	52.70% (N=39)	60.81% (N=45)
Hypothyroidism N=22	18.18% (N=4)	9.09% (N=2)	13.64% (N=3)	18.18% (N=4)	**54.55% (N=12)**	18.18% (N=4)	50.00% (N=11)
Bronchial asthma N=22	22.73% (N=5)	22.73% (N=5)	22.73% (N=5)	22.73% (N=5)	31.82% (N=7)	27.27% (N=6)	**36.36% (N=8)**
Dyslipidemia N=10	50.00% (N=5)	50.00% (N=5)	60.00% (N=6)	60.00% (N=6)	60.00% (N=6)	60.00% (N=6)	**70.00% (N=7)**

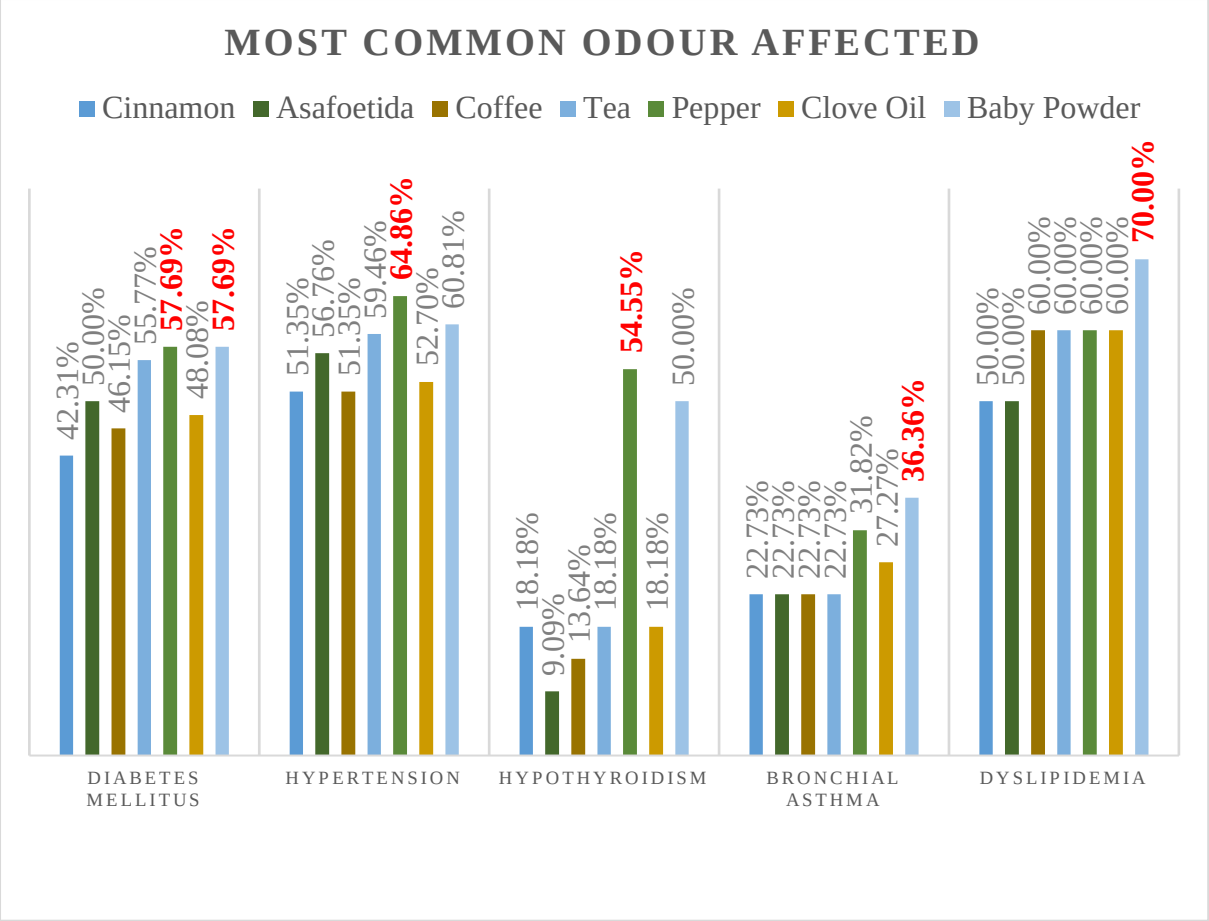


Figure 24 - Odour vs comorbidities

Prevalence of nasal diseases:

The patients within the study population had the following nasal diseases namely, allergic rhinitis (AR), chronic rhinosinusitis without nasal polyposis (CRS without NP), chronic rhinosinusitis with nasal polyposis and sinonasal polyposis (CRS with NP + SNP), post traumatic anosmia, atrophic rhinitis and idiopathic olfactory loss. Post surgical patients were not included in the study. The most common nasal disease found among them was allergic rhinitis.

Table 9: Prevalence of nasal diseases

Sl No.	Nasal disease	No.	%
1	AR	212	44.35%
2	CRS without NP	32	6.69%
3	CRS with NP + SNP	96	20.08%
4	Post traumatic anosmia	26	5.44%
5	Atrophic rhinitis	20	4.18%
6	Idiopathic	92	19.25%

(Post *viral and post FESS patients within the 490 have been taken out as their numbers were very small and analysis could not be done. Hence the total number among the nasal diseases is 478.)*

Prevalence of nasal diseases vs types of olfactory dysfunction:

The patients with allergic rhinitis were more in number as compared to other types of nasal diseases and they showed anosmia and moderate hyposmia type of olfactory dysfunction. Among the patients who had chronic rhinosinusitis, post traumatic smell loss, atrophic rhinitis and idiopathic loss of smell anosmia was the predominant type of olfactory dysfunction.

Table 10: Prevalence of nasal diseases vs types of olfactory dysfunction

Types	Anosmia	Severe hyposmia	Moderate hyposmia	Mild hyposmia
AR N=212	30.66% N=65	23.11% N=49	26.89% N=57	18.87% N=40
CRS without NP N=32	53.13% N=17	34.38% N=11	6.25% N=2	6.25% N=2
CRS with NP + SNP N=96	58.33% N=56	9.38% N=9	13.54% N=13	15.63% N=15
Post traumatic anosmia N=26	92.31% N=24	0.00% N=0	7.69% N=2	0.00% N=0
Atrophic rhinitis N=20	90.00% N=18	10.00% N=2	0.00% N=0	0.00% N=0
Idiopathic N=92	46.74% N=43	33.70% N=31	11.96% N=11	4.35% N=4

(Note: No. and % of Normosmia is not shown here)

Most common odour affected in various nasal diseases

While analysing the specific smell loss in every nasal disease group, pepper was the least perceived smell among the allergic rhinitis, CRS without nasal polyposis, CRS with nasal polyposis and idiopathic anosmia patients.

Table 11: Odour vs nasal diseases

Nasal diseases	Cinnamon	Asafoetida	Coffee	Tea	Pepper	Clove Oil	Baby Powder
AR	28.30%	29.25%	33.02%	37.26%	**50.94%**	32.08%	42.45%
CRS without NP	46.88%	53.13%	43.75%	62.50%	**71.88%**	56.25%	65.63%
CRS with NP + SNP	58.33%	56.25%	55.21%	56.25%	**64.58%**	59.38%	62.50%
Post traumatic anosmia	84.62%	80.77%	92.31%	92.31%	92.31%	92.31%	88.46%
Atrophic rhinitis	90.00%	**95.00%**	90.00%	90.00%	90.00%	90.00%	**95.00%**
Idiopathic	50.00%	55.43%	50.00%	50.00%	**73.91%**	50.00%	66.30%

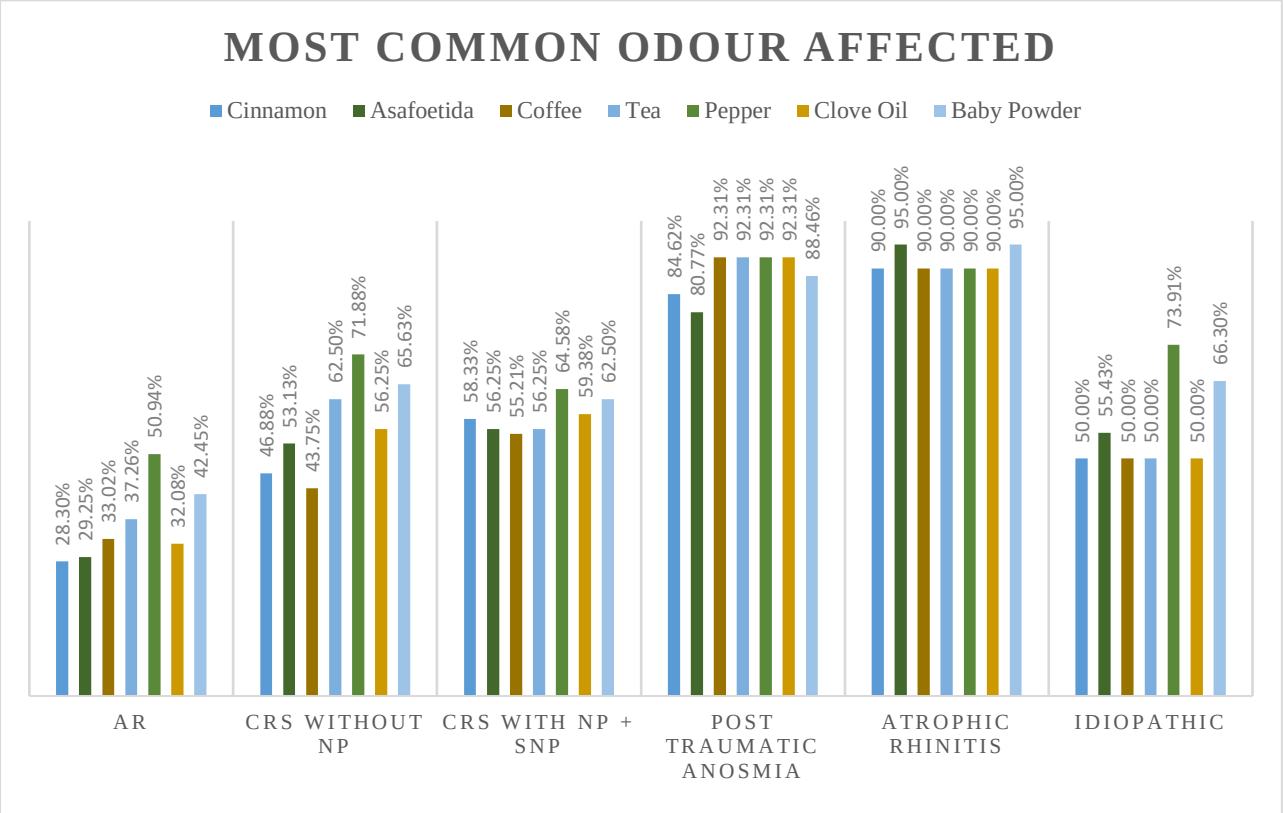


Figure 25 - Most common odour affected in nasal diseases

Odour identification scores in nasal diseases:

Among the study population, patients with chronic rhinosinusitis with and without polyposis, post traumatic anosmia, atrophic rhinitis and idiopathic anosmia had an odour identification score of 0, whereas it was 7 among patients with allergic rhinitis.

Table 12: Odour identification scores in nasal diseases

Odour Identification	AR N=212		CRS without NP N=32		CRS with NP + SNP N=96		Post traumatic anosmia N=26		Atrophic rhinitis N=20		Idiopathic N=92	
	No	%	No	%	No	%	No	%	No	%	No	%
0	41	19.34 %	14	**43.75 %**	47	**48.96%**	20	**76.92%**	18	**90.00 %**	43	**46.74 %**
1	9	4.25 %	0	0.00%	4	4.17%	2	7.69 %	0	0.00 %	0	0.00 %
2	13	6.13 %	0	0.00%	5	5.21%	2	7.69 %	0	0.00 %	0	0.00 %
3	8	3.77 %	4	12.50%	1	1.04%	0	0.00 %	0	0.00 %	1	1.09 %
4	13	6.13 %	2	6.25%	0	0.00%	0	0.00 %	0	0.00 %	4	4.35 %
5	16	7.55 %	2	6.25%	4	4.17%	0	0.00 %	0	0.00 %	19	20.65 %
6	28	13.21 %	4	12.50%	7	7.29%	0	0.00 %	2	10.00 %	9	9.78 %
7	84	**39.62 %**	6	18.75%	28	29.17%	2	7.69 %	0	0.00 %	16	17.39 %

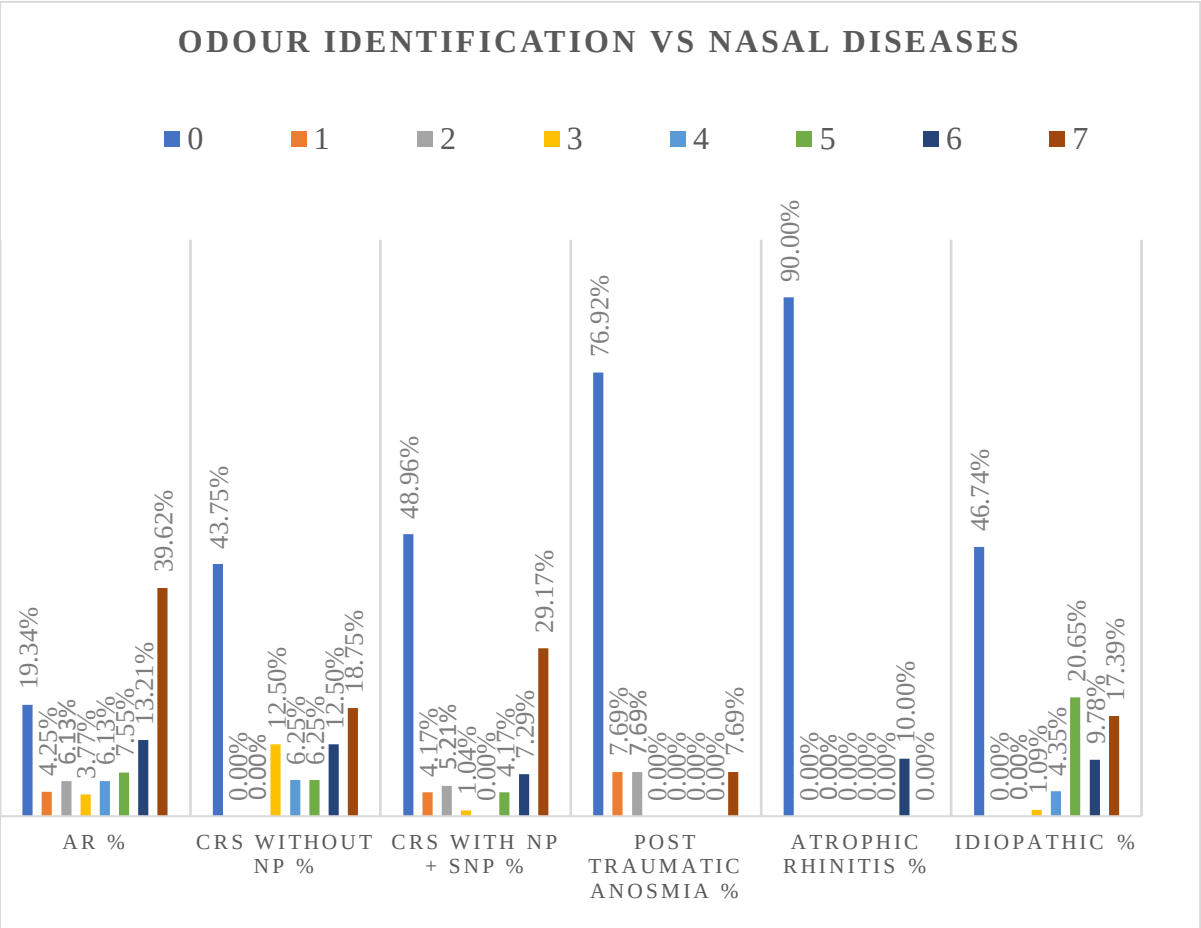


Figure 26 - Odour identification vs nasal diseases

Olfactory threshold scores in nasal diseases:

The patients in the study cohort had a predominant olfactory threshold score of zero among all the nasal diseases.

Table 13: Olfactory threshold scores in nasal diseases

Olfactory Threshold	AR N=212		CRS without NP N=32		CRS with NP + SNP N=96		Post traumatic anosmia N=26		Atrophic rhinitis N=20		Idiopathic N=92	
	N	%	N	%	N	%	N	%	N	%	N	%
0	90	**42.86 %**	24	**75.00 %**	58	**61.05 %**	24	**92.31 %**	18	**90.00 %**	64	**71.11 %**
1	21	10.00 %	4	12.50 %	13	13.68 %	0	0.00%	1	5.00%	4	4.44%
2	43	20.48 %	2	6.25%	4	4.21%	2	7.69%	0	0.00%	16	17.78 %
3	45	21.43 %	2	6.25%	14	14.74 %	0	0.00%	1	5.00%	4	4.44%
4	9	4.29%	0	0.00%	3	3.16%	0	0.00%	0	0.00%	0	0.00%
5	2	0.95%	0	0.00%	3	3.16%	0	0.00%	0	0.00%	2	2.22%
6	0	0.00%	0	0.00%	0	0.00%	0	0.00%	0	0.00%	0	0.00%

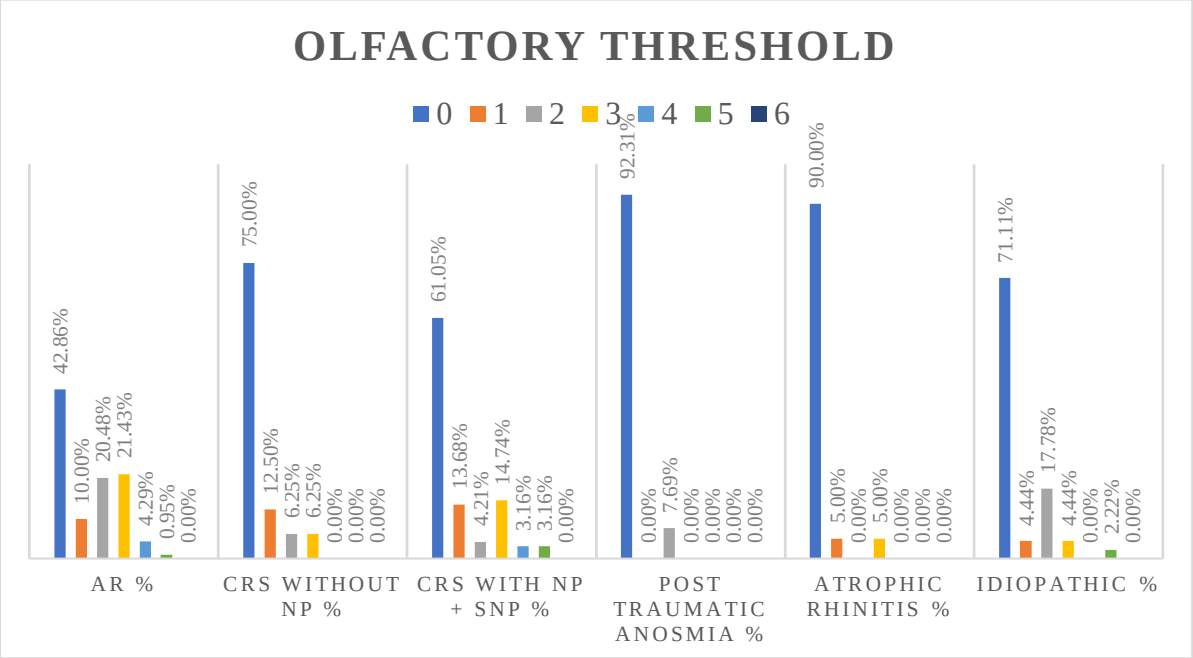


Figure 27 - Olfactory threshold in nasal diseases

DISCUSSION

DISCUSSION

Olfactory dysfunction has a worldwide incidence of 1.4% adult population. It is usually disregarded by the clinicians and the patients visit multiple physicians to get the problem addressed. Most of the patients who complain of taste disturbance also have a coexisting olfactory dysfunction. The evaluation and timely management of olfactory dysfunction is very important as it interferes with the quality of life, appetite and psychological wellbeing (1).

A retrospective analysis of all the patients who underwent the CCCRC (Connecticut Chemosensory Clinical Research Centre) olfaction test within the study period from January 2018 to March 2020 were done. Based on the composite score calculated from the results of the 2 components of the CCCRC test, a diagnosis of anosmia, severe hyposmia, moderate hyposmia, mild hyposmia and normosmia was made. Patients who had olfactory dysfunction were included in the study and those with normal olfaction were excluded. Patients who had undergone transnasal endoscopic surgeries were excluded for the sake of eliminating bias in the results owing to the surgical intervention.

Though there were 245 patients satisfying the inclusion and exclusion criteria, as there was different grade of olfactory dysfunction for each nostril in every patient, the total sample size was taken as 490 nostrils. In our study, we collected data including demography, endoscopic findings, clinical findings and olfactory test scores.

Among the study population, there were151 males (61.63%) and 94 females (38.37%) and all four types of olfactory dysfunction were seen predominantly in men

compared to women. Richard L Doty has compiled the epidemiological statistics of olfactory dysfunction from 16 other studies with a wide range of age population. The prevalence of olfactory loss was more pronounced in the women than men in 11 out of 16 studies. He attributed the female preponderance to their involvement in cooking and caregiving (42). This is in contrast to our cohort where the male gender is more affected than the women.

In our study, the age of the patients ranged from 18 to 72 years. The majority of the patients in all four types of olfactory dysfunction belonged to the 18-45 age group. Doty, et al., has studied the age-related changes in chemosensory functions like taste and smell. He assessed the olfaction by the UPSIT (University of Pennsylvania Smell Identification Test) in a group of general population with age ranging from 5 to 90 years. It was found that people who are 60 years and above have decline in olfactory perception. He has attributed the incidence to three reasons: 1. Exposure to air pollution, bacteria, cigarette smoke and viruses causing damage to olfactory epithelium, 2. Reduced receptor cell regeneration following damage in the elderly, 3. Reduced size and less number of foramina in cribriform plate blocking the passage of olfactory receptor cell axons to the brain through the nasal cavity (43). The comparatively lower mean age (41 years) in our study population could be because this was not a community study and only done among patients who sought medical care for the olfactory loss.

There were many nasal diseases among the study population, namely, allergic rhinitis, chronic rhinosinusitis without polyposis, chronic rhinosinusitis with polyposis, atrophic rhinitis and post traumatic anosmia. Out of all the nasal diseases, allergic rhinitis was seen in 44.35%, making it the most predominant.

Patients with allergic rhinitis had anosmia followed by moderate hyposmia as the most common olfactory dysfunction, whereas chronic rhinosinusitis with or without polyposis patients showed anosmia followed by severe hyposmia. While performing the odor identification test, we found that pepper was least perceived among both allergic rhinitis and chronic rhinosinusitis patients.

In 1999, Andrea, et al., studied about the correlation between olfactory loss among the patients with allergic rhinitis and chronic rhinosinusitis. The patients underwent endoscopic nasal examination, olfactory testing (butanol threshold and odor identification using common household items like coffee, baby powder, peanut butter and chocolate) and computed tomography to look for olfactory clefts. It was found that the incidence and severity of olfactory loss is more among the allergic rhinitis patients with rhinosinusitis as they are prone for respiratory infections which can cause olfactory epithelial loss. However, they have not mentioned the specific odor loss with respect to allergic rhinitis and chronic rhinosinusitis (30). In another study by Jamie, et al., in 2008, on patients with pre-existing chronic rhinosinusitis, age more than 65 years, nasal polyposis, asthma and smoking had significant olfactory dysfunction. But allergic rhinitis was not a major contributor for causing smell loss (44).

In our cohort, 19% of patients had idiopathic anosmia which can be attributed to olfactory loss following viral upper respiratory tract infections. In a compilation by Allen.M. Seiden of postviral olfactory loss statistics, he has mentioned that there was 18.6% of patients in the Connecticut Chemosensory Clinical Research Centre who had postviral anosmia following upper respiratory tract infections. As the pathophysiology behind the postviral loss is unclear, he suggests that it can be due to destruction or

degeneration of the olfactory receptor cells either in the central or peripheral pathways following a viral infection (23).

The comorbidities prevalent among the study population were diabetes mellitus, hypertension, hypothyroidism, bronchial asthma and dyslipidemia. The most common comorbidity was found to be systemic hypertension followed by diabetes mellitus. Anosmia was seen predominantly among the diabetics, hypertensives and dyslipidemia patients, whereas severe hyposmia was the most common type of olfactory dysfunction seen in hypothyroid patients and moderate hyposmia in asthmatics. The odour identification revealed that the diabetics had difficulty perceiving pepper and baby powder. Among the hypertensives and patients with hypothyroidism, it was pepper that was the least perceived. The baby powder was less perceived by the patients who had bronchial asthma and dyslipidemia.

In our study, men were affected more than the women among the diabetics. Ruth, et al., studied 111 diabetic patients and assessed their olfaction by Confusion Matrix test and found that the olfactory dysfunction is related to increasing age, presence of macrovascular disease, smoking and male sex (45). In another study done by Gouveri, et al., olfactory dysfunction was analysed among 154 adults with diabetes. He concluded that the diabetic peripheral neuropathy and retinopathy are increasingly associated with olfactory loss than diabetes alone (28). In our study, the evidence of diabetic peripheral neuropathy and retinopathy were lacking as it was a retrospective cohort. Hence the association between the smell loss and the diabetic neuropathy was not noted.

In our study, 52% of the hypertensive patients had anosmia and odor identification test revealed that pepper was the least perceived odor among the seven odors. Being a retrospective cohort, our study has not assessed the onset of olfactory loss in relation to the onset of systemic hypertension. A prospective study done in China among 5190 adults by Liu, et al., studied the relationship between the rising blood pressures and smell taste perception over a period of two years. Blood pressures were measured at the beginning of the study and after two years while olfaction was also assessed with the help of a questionnaire. It was found that the number of people who had high blood pressures at the end of two years also showed reduced perception of smell and taste overtime. The systolic blood pressure as well as the mean arterial pressure was found to be higher in people with chemosensory dysfunction (46). In this study the severity of olfactory dysfunction such as anosmia or hyposmia and loss of specific odors were not assessed because subjective olfaction test was not performed among the study population.

In our cohort, the bronchial asthma patients showed reduced smell perception and majority of them fell in the moderate hyposmia type of olfactory dysfunction. In a retrospective study conducted in Japan by Koichiro, et al., among 56 bronchial asthma patients, the olfaction was assessed by self-administered odor questionnaire (SAOQ). The patients with bronchial asthma and eosinophilic chronic rhinosinusitis had significant low SAOQ scores indicating olfactory dysfunction in those patients. But the severity of olfactory dysfunction was not identified in that study (47).

The two components of the CCCRC test were analysed with the different grades of olfactory dysfunction. The n-butanol threshold has scores from 0 to 6 and odour

identification scores range from 0 to 7. The patients with anosmia and severe hyposmia had a predominant olfactory threshold score of zero. Among patients with moderate hyposmia, a majority had a threshold score of 2 followed by score 1. In the mild hyposmia group, the dominant score was 3. In the odour identification component, majority of patients with anosmia had an odour identification score of 0, whereas it was 6 followed by 5 within severe hyposmia group. In the moderate and mild hyposmia population the most common score was 7. The odour identification scores were higher even among severe hyposmia, moderate hyposmia and mild hyposmia groups. Therefore, the novel finding from our study is that the odour identification component gives a majority weightage in determining the composite score thereby the type of olfactory dysfunction. Our study is the first one to arrive at a conclusion as there are no similar studies to compare this analysis.

The limitation of our study is that being a retrospective study, the level of evidence is low when compared to the prospective studies.

CONCLUSION

CONCLUSION

Olfactory dysfunction is a debilitating health problem which is usually undiagnosed. Patients who are young most often present with complaints of smell loss, whereas elderly population tend to live with the problem. All grades of olfactory dysfunction are predominantly noted among the male population. People in the fourth to sixth decade has the highest incidence of smell loss. Anosmia was seen predominantly among the diabetics, hypertensives and dyslipidemia patients, whereas severe hyposmia was the most common type of olfactory dysfunction seen in hypothyroid patients and moderate hyposmia in asthmatics. Pepper was the least perceived and most probably the early odor to be lost in patients with diabetes, hypertension, hypothyroidism. Baby powder was not perceived by the patients with bronchial asthma and dyslipidemia.

Anosmia and moderate hyposmia were seen among the allergic rhinitis patients, whereas in those with chronic rhinosinusitis, post traumatic smell loss, atrophic rhinitis and idiopathic loss of smell, anosmia was the predominant type of olfactory dysfunction. Pepper was the least perceived smell among the allergic rhinitis, CRS with and without nasal polyposis, and idiopathic anosmia patients. In patients with atrophic rhinitis, asafoetida and baby powder were affected.

On analysing the components of the CCCRC test against the types of olfactory dysfunction, it was seen that the odour identification component gives a majority weightage in determining the composite score thereby the type of olfactory dysfunction.

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ANNEXURES

ANNEXURES

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DATA SHEET

Sl.No.	Age	Sex	Group	Onset of loss of smell	Duration (Months)	History of prior viral illness	Comorbidities	Diabetes	Hypertension	Hypothyroidism	Bronchial asthma	Dyslipidemia	AR	CRS	Others	Hb	RBS	Creatinine	RNE	Diagnosis	Nasal Disease	Test	Odour identification test									Score			Types of olfactory dysfunction
																						Olfactory threshold	Cinnamon	Asafoetida	Coffee	Tea	Pepper	Clove oil	Baby powder	Total correct	Eucalyptus (Trigeminal)	Olfactory threshold	Odour identification	Composite score	
1	31	F	3	2	48			No	No	No	No	No	Yes	No					AR, DNS to L with spur to R	AR, Anosmia	AR	2	1	1	1	1	1	1	1	7	1	2	7	4.5	Moderate
2	46	M	1	2	4			No	No	No	No	No	No	No		15	101	0.96	Sharp spur to R	Anosmia	Idopathic	0	0	0	0	0	0	0	0	0	1	0	0	0	Anosmia
3	42	F	4	2	240		Hypothyroid	No	No	Yes	No	No	Yes	No			118	0.73	AR	AR, Anosmia	AR	3	1	1	1	1	1	1	1	7	1	3	7	5	Mild
4	53	F	1	2	120		SHTN	No	Yes	No	No	No	No	Yes		13	129	0.66	CRS with fungal ball L	CRS	CRS wiyhout NP	0	0	0	0	0	0	0	0	0	1	0	0	0	Anosmia
5	31	F	3	2	36			No	No	No	No	No	Yes	No		12.7	129	0.76	AR, DNS with spur to L	AR, Anosmia	AR	1	1	1	1	1	1	1	1	7	1	1	7	4	Moderate
6	52	M	3	0			SHTN	No	Yes	No	No	No	No	No		14.1	118	0.87	R SNP	SNP	SNP	1	1	1	1	1	1	1	1	7	1	1	7	4	Moderate
7	23	M	2	0				No	No	No	No	No	Yes	No		15.3	118	0.88	AR, DNS with spur to R	AR	AR	1	1	1	1	1	0	1	1	6	1	1	6	3.5	Severe
8	33	F	3	0				No	No	No	No	No	Yes	No		13	80	0.6	AR, DNS to R	AR	AR	2	1	1	1	1	1	1	1	7	1	2	7	4.5	Moderate
9	25	M	2	2	5			No	No	No	No	No	Yes	No		15.6	89	0.96	AR	AR, Anosmia	AR	0	1	1	0	1	1	1	1	6	1	0	6	3	Severe
10	37	M	1	0				No	No	No	No	No	Yes	No		15.5	87		AR, B/L ETD, DNS to R, POST OP	AR	AR	1	1	0	0	0	0	0	0	1	1	1	1	1	Anosmia
11	33	M	4	0			SHTN	No	Yes	No	No	No	Yes	No		14.9	396	0.87	AR, DNS with spur to R	AR	AR	4	1	1	1	1	0	1	1	6	1	4	6	5	Mild
12	22	M	4	0				No	No	No	No	No	Yes	No		15.9	86	0.88	AR, DNS to R with spur to L	AR	AR	5	1	1	1	0	1	0	1	5	1	5	5	5	Mild
13	36	M	2	2	84			No	No	No	No	No	No	No		13.8	104	1.04	Atrophic rhinitis	Atrophic rhinitis	Atrophic rhinitis	0	1	1	1	1	1	1	0	6	1	0	6	3	Severe
14	50	F	4	0			Hypothyroid	No	No	Yes	No	No	Yes	No		11.9	159	0.73	AR	AR	AR	3	1	1	1	1	1	1	1	7	1	3	7	5	Mild
15	21	F	4	0				No	No	No	No	No	Yes	No		10.6	96	0.48	AR	AR, Anosmia	AR	3	1	1	1	1	1	1	1	7	1	3	7	5	Mild
16	23	M	3	2	36			No	No	No	No	No	Yes	No		16.2	84	0.77	AR	AR, Anosmia	AR	2	1	1	1	1	0	1	1	6	1	2	6	4	Moderate
17	30	F	3	2	36			No	No	No	No	No	Yes	No		11.6	89	0.61	AR, DNS to L	AR, Anosmia	AR	1	1	1	1	1	1	1	1	7	1	1	7	4	Moderate
18	33	M	1	1	144	RTA		No	No	No	No	No	No	No		13.9	93	0.8	SNP	Post traumatic Anosmia	Post traumatic Anosmia	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
19	22	M	3	0				No	No	No	No	No	Yes	No		15.2	88	0.79	AR	AR	AR	2	1	1	1	1	1	1	1	7	1	2	7	4.5	Moderate
20	42	M	1	1	30	RTA		No	No	No	No	No	No	No					High DNS to R	Traumatic Anosmia	Post traumatic Anosmia	0	1	1	0	0	0	0	0	2	1	0	2	1	Anosmia
21	33	F	2	2	30		BA	No	No	No	Yes	No	Yes	No		13.1	104	0.64	AR	AR, Anosmia	AR	0	1	1	1	1	1	0	0	5	1	0	5	2.5	Severe
22	50	F	1	2	72			No	No	No	No	No	No	No		14.6	94	0.72	Spur to L	Anosmia	Idopathic	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
23	36	M	4	2	180			No	No	No	No	No	Yes	No		15.3	103	0.79	AR, DNS to L with spur, R concha	AR, Anosmia	AR	3	1	1	1	1	1	1	1	7	1	3	7	5	Mild
24	36	M	4	0			SHTN	No	Yes	No	No	No	No	Yes		15.4	133	0.95	CRS with NP	CRS with NP	CRS with NP	3	1	1	1	1	1	1	1	7	1	3	7	5	Mild
25	26	M	2	0				No	No	No	No	No	Yes	Yes		12.8	124	0.89	AR, CRS	AR, CRS	AR	3	1	1	1	0	0	1	0	4	1	3	4	3.5	Severe
26	58	F	4	0				No	No	No	No	No	Yes	No		12.7	89	0.6	AR	AR	AR	3	1	1	1	1	1	1	1	7	1	3	7	5	Mild
27	47	F	4	0				No	No	No	No	No	Yes	No		11.6	80	0.66	AR	AR	AR	3	1	1	1	1	1	1	1	7	1	3	7	5	Mild
28	31	F	1	2	48			No	No	No	No	No	No	Yes		13.5	90	0.6	CRS with NP	CRS with NP	CRS with NP	0	0	0	0	0	0	0	0	0	1	0	0	0	Anosmia
29	45	M	4	0			SHTN, Dyslipidemia	No	Yes	No	No	Yes	Yes	No		15.1	163	0.74	AR, DNS to L	AR	AR	3	1	1	1	1	1	1	1	7	1	3	7	5	Mild
30	38	M	1	1	24	RTA		No	No	No	No	No	No	No					DNS to R	Traumatic Anosmia	Post traumatic Anosmia	0	0	1	0	0	0	0	0	1	1	0	1	0.5	Anosmia
31	30	M	2	0				No	No	No	No	No	Yes	No		14.9	100	0.82	R SNP, AR	SNP, AR	SNP	1	1	1	1	0	0	0	0	3	1	1	3	2	Severe
32	20	M	2	0				No	No	No	No	No	Yes	No		15.2	88	0.72	High DNS to R, AR	AR	AR	2	1	1	1	1	0	0	0	4	1	2	4	3	Severe
33	42	M	3	0				No	No	No	No	No	Yes	No		15	110	0.92	AR, DNS to R	AR	AR	2	1	1	1	1	1	1	1	7	1	2	7	4.5	Moderate
34	38	F	1	0				No	No	No	No	No	Yes	No		13.4	92	0.62	DNS to R, AR	AR	AR	0	0	1	0	0	0	0	0	1	1	0	1	0.5	Anosmia
35	47	M	1	2	120			No	No	No	No	No	No	No					Post op status - FESS	CRS with NP	CRS with NP	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
36	47	M	2	2	72			No	No	No	No	No	No	No		14	102	0.95	DNS to R	Anosmia	Idopathic	0	1	1	1	1	0	1	1	6	1	0	6	3	Severe
37	32	F	3	0				No	No	No	No	No	No	No		11	104	0.55	B/L ITH	AR	AR	3	1	1	1	1	0	1	1	6	1	3	6	4.5	Moderate
38	41	F	1	0				No	No	No	No	No	No	Yes		13.2		0.48	CRS with NP	CRS with NP	CRS with NP	0	0	1	0	0	0	0	0	1	1	0	1	0.5	Anosmia
39	30	M	2	0				No	No	No	No	No	Yes	No		13.5	90	0.86	AR, DNS to L	AR	AR	1	1	1	0	1	0	1	0	4	1	1	4	2.5	Severe
40	55	F	1	0				No	No	No	No	No	Yes	No		10.3	89	0.48	AR	AR	AR	0	0	1	0	0	0	0	0	1	1	0	1	0.5	Anosmia
41	37	M	4	0				No	No	No	No	No	Yes	No		13.3	93	1.14	AR, DNS to L with spur to L	AR	AR	3	1	1	1	1	1	1	1	7	1	3	7	5	Mild
42	34	M	3	2	84		Dyslipidemia	No	No	No	No	Yes	Yes	No		13.6	110	0.77	AR, DNS with spur to L	AR, Anosmia	AR	2	1	1	1	1	1	1	1	7	1	2	7	4.5	Moderate
43	33	F	2	0			Hypothyroid	No	No	Yes	No	No	No	No		12.4	110	0.62	Gross DNS to R	Anosmia	Idopathic	2	1	1	1	1	0	1	0	5	1	2	5	3.5	Severe
44	32	M	1	0				No	No	No	No	No	No	No	Post op FESS	15.6	94	0.86	B/L SNP, septal perforation	Recurrent SNP	SNP	0	0	0	0	0	0	0	0	0	1	0	0	0	Anosmia
45	44	M	4	2	96			No	No	No	No	No	No	Yes		13.9	94	0.86	CRS without NP, spur to L	CRS without NP	CRS without NP	3	1	1	1	1	1	1	1	7	1	3	7	5	Mild
46	39	M	1	2	240			No	No	No	No	No	No	No		12.2	92	0.69	Atrophic rhinitis	Atrophic rhinitis, Anosmia	Atrophic rhinitis	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
47	38	M	2	2	36			No	No	No	No	No	Yes	No		13.9	102	1.06	AR, B/L ETD, DNS with spur	AR, Anosmia	AR	1	1	1	0	0	0	1	0	3	1	1	3	2	Severe
48	49	F	4	0				No	No	No	No	No	No	Yes		13.1	92	0.53	CRS with NP	CRS with NP	CRS with NP	3	1	1	1	1	1	1	1	7	1	3	7	5	Mild
49	49	F	1	1	120			No	No	No	No	No	No	No		11.7	106		Normal	Anosmia	Idopathic	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
50	45	F	1	2	48			No	No	No	No	No	No	Yes		11.5	94	0.77	CRS with NP, DNS to R	CRS with NP	CRS with NP	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
51	34	F	2	2	36			No	No	No	No	No	Yes	No		12	126	0.77	AR	AR, Anosmia	AR	0	1	1	1	1	1	1	1	7	1	0	7	3.5	Severe
52	24	F	1	2	60			No	No	No	No	No	No	No		15.8	78	0.53	B/L sphenoethmoidal polyp	B/L sphenoethmoidal polyp	SNP	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
53	59	F	2	2	36		Hypothyroid	No	No	Yes	No	No	Yes	No		12.1	95	0.85	AR, DNS to L	AR, Anosmia	AR	2	0	1	1	1	0	0	0	3	1	2	3	2.5	Severe
54	52	M	2	0				No	No	No	No	No	Yes	No		14.5	89	0.89	AR, B/L ITH	AR	AR	0	1	1	1	1	0	1	1	6	1	0	6	3	Severe
55	62	M	3	2	36			No	No	No	No	No	No	No		13.1	114	0.89	High DNS to R	Anosmia	Idopathic	1	1	1	1	1	1	1	1	7	1	1	7	4	Moderate
56	23	M	1	2	72			No	No	No	No	No	No	Yes		16.2		0.97	CRS, DNS to R	CRS	CRS without NP	0	0	0	1	1	0	1	0	3	1	0	3	1.5	Anosmia
57	15	M	4	0				No	No	No	No	No	No	No		13.7	108	0.98	L recurrent AC polyp	L recurrent AC polyp	SNP	3	1	1	1	1	1	1	1	7	1	3	7	5	Mild
58	45	M	1	2	120			No	No	No	No	No	No	Yes		14.2	123	0.79	CRS with NP	CRS with NP	CRS with NP	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
59	47	F	1	0				No	No	No	No	No	No	Yes		12.3	123	0.67	CRS	CRS	CRS without NP	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
60	46	M	4	0				No	No	No	No	No	Yes	No		11.9	161	1.24	AR, high DNS to R	AR	AR	3	1	1	1	1	1	1	1	7	1	3	7	5	Mild
61	40	M	1	2	180			No	No	No	No	No	No	Yes		15.8	96	1.11	CRS with NP	CRS with NP	CRS with NP	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
62	46	M	4	0			DM	Yes	No	No	No	No	Yes	No		13.1	143	0.76	AR	AR	AR	3	1	1	1	1	1	1	1	7	1	3	7	5	Mild
63	54	M	1	1	120	RTA		No	No	No	No	No	Yes	No		15.2	145	1.21	AR	Post traumatic Anosmia	Post traumatic Anosmia	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
64	45	M	1	1	4	Fever		No	No	No	No	No	No	No		10.6	96	0.73	Spur to L	Post viral Anosmia	Post viral Anosmia	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
65	63	M	1	2				No	No	No	No	No	Yes	No		12.7	121	0.96	AR	AR, Anosmia	AR	0	1	1	0	0	0	1	0	3	1	0	3	1.5	Anosmia
66	26	M	3	0				No	No	No	No	No	Yes	No		13.6	89	0.59	AR, DNS with spur to L	AR	AR	3	1	1	0	1	1	1	1	6	1	3	6	4.5	Moderate
67	49	F	2	2	5			No	No	No	No	No	Yes	No					AR	AR, Anosmia	AR	0	1	1	1	0	0	1	0	4	1	0	4	2	Severe
68	25	F	1	0				No	No	No	No	No	No	Yes		12.7	85	0.2	CRS with NP	CRS with NP	CRS with NP	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
69	26	M	2	0				No	No	No	No	No	Yes	No		12.1	90	0.63	AR	AR	AR	2	1	0	0	1	0	1	1	4	1	2	4	3	Severe
70	54	F	4	0			DM	Yes	No	No	No	No	Yes	No		9.7	223	1.53	AR	AR	AR	3	1	1	1	1	1	1	1	7	1	3	7	5	Mild
71	41	F	1	2	7	URTI		No	No	No	No	No	Yes	No		11.9	96	0.75	AR	Post viral Anosmia	Post viral Anosmia	0	1	1	0	0	0	0	1	3	1	0	3	1.5	Anosmia
72	51	M	1	2	36		DM	Yes	No	No	No	No	No	No		12.5	149	1.32			Idopathic	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
73	40	M	1	0			Dyslipidemia, DM	Yes	No	No	No	Yes	Yes	No		15.5	93	0.92	AR	AR, Anosmia	AR	0	1	1	0	0	0	0	0	2	1	0	2	1	Anosmia
74	24	F	2	2	7			No	No	No	No	No	No	No	L COM squamosal inactive	13.8	97	0.53	DNS to R	Anosmia	Idopathic	0	1	1	1	1	0	1	0	5	1	0	5	2.5	Severe
75	47	M	3	0				No	No	No	No	No	Yes	No		14	102	1.06	AR	AR	AR	2	1	1	1	1	1	1	1	7	1	2	7	4.5	Moderate
76	49	M	1	2	60		SHTN	No	Yes	No	No	No	No	Yes		8.4	110	0.98	CRS with NP	CRS with NP	CRS with NP	0	0	0	0	0	0	0	0	0	1	0	0	0	Anosmia
77	40	F	2	2	48			No	No	No	No	No	No	No		12.9	96	0.59	DNS to R	AR, Anosmia	AR	0	1	1	1	1	1	1	1	7	1	0	7	3.5	Severe
78	54	M	1	2	84			No	No	No	No	No	No	Yes		11.3	96	0.83	CRS with NP, DNS to R	CRS with NP	CRS with NP	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
79	60	M	1	2	36			No	No	No	No	No	No	Yes		12.9	125	0.66	CRS with NP	CRS with NP	CRS with NP	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
80	45	M	1	2	6	URTI		No	No	No	No	No	Yes	No		13.6	93	0.82	AR	AR, Anosmia	AR	0	0	0	0	0	0	1	0	1	1	0	1	0.5	Anosmia
81	32	F	2	0				No	No	No	No	No	Yes	No		11.4	100	0.56	AR, DNS with spur to R	AR	AR	0	1	1	1	1	1	1	1	7	1	0	7	3.5	Severe
82	28	M	3	2	12			No	No	No	No	No	No	No		14.9	107	0.98	Sinusitis	Sinusitis, Anosmia	CRS without NP	2	1	1	1	1	1	0	1	6	1	2	6	4	Moderate
83	58	F	1	2	120		SHTN	No	Yes	No	No	No	No	No		14.4	107	0.53	High DNS to L	Anosmia	Idopathic	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
84	42	F	1	2	96			No	No	No	No	No	Yes	No		13.9	146	0.61	AR	AR, Anosmia	AR	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
85	59	M	1	2	120		SHTN	No	Yes	No	No	No	No	Yes		11.2	148	1.11	CRS with NP	CRS with NP	CRS with NP	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
86	66	M	1	2	36		SHTN	No	Yes	No	No	No	No	No		10.1	112	1.29	Atrophic rhinitis	Atrophic rhinitis, Anosmia	Atrophic rhinitis	0	0	0	0	0	0	0	0	0	1	0	0	0	Anosmia
87	34	M	1	2	180	RTA		No	No	No	No	No	No	No		14.1	101	0.73	DNS with spur to L	Post traumatic Anosmia	Post traumatic Anosmia	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
88	43	M	1	2	4			No	No	No	No	No	No	No				0.81	Sharp spur to L	Anosmia	Idopathic	2	0	0	0	0	0	0	0	0	0	2	0	1	Anosmia
89	61	M	4	0			DM, SHTN, IHD	Yes	Yes	No	No	No	Yes	No		12.9	142	1	AR, DNS with spur to R	AR	AR	3	1	1	1	1	1	1	1	7	1	3	7	5	Mild
90	39	M	1	2	12		DM	Yes	No	No	No	No	Yes	No		15.5	251	0.88	AR, DNS with spur to L	AR, Anosmia	AR	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
91	36	M	1	2	120		SHTN	No	Yes	No	No	No	Yes	No		15.8	97	0.73	AR	AR, Anosmia	AR	0	0	0	1	0	0	0	1	2	1	0	2	1	Anosmia
92	36	F	1	1	1	RTA		No	No	No	No	No	No	No					Normal	Post traumatic Anosmia	Post traumatic Anosmia	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
93	43	M	2	2	72			No	No	No	No	No	No	Yes		16.7	108	0.67	CRS	CRS	CRS without NP	1	1	1	1	0	0	0	0	3	1	1	3	2	Severe
94	38	F	1	2	6		DM	Yes	No	No	No	No	No	Yes		12	331	0.71	CRS with NP	CRS with NP	CRS with NP	0	0	0	0	1	0	0	0	1	1	0	1	0.5	Anosmia
95	41	M	1	2	72		SHTN	No	Yes	No	No	No	No	No		13.9	106	0.84	R SNP	SNP	SNP	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
96	55	M	3	0			DM, SHTN	Yes	Yes	No	No	No	Yes	No		12.9	86	1.26	AR	AR	AR	2	1	1	1	1	1	1	1	7	1	2	7	4.5	Moderate
97	65	F	1	2	12		DM, SHTN	Yes	Yes	No	No	No	No	Yes		12.8	99	0.87	CRS with NP	CRS with NP	CRS with NP	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
98	49	M	1	2	36			No	No	No	No	No	No	Yes		14.6	109	1.05	CRS with NP	CRS with NP	CRS with NP	1	0	0	1	0	0	0	0	1	1	1	1	1	Anosmia
99	23	M	4	1	36	Chicken pox		No	No	No	No	No	Yes	No		15.8	89	0.91	AR	AR	AR	5	0	0	1	1	1	1	1	5	1	5	5	5	Mild
100	38	M	4	2	18			No	No	No	No	No	No	Yes		13.9	91	0.98	CRS with NP	CRS with NP	CRS with NP	4	0	1	1	1	1	1	1	6	1	4	6	5	Mild
101	32	F	4	0				No	No	No	No	No	No	Yes		10.9		0.77	CRS with NP	CRS with NP, AR	CRS with NP	3	1	1	1	1	1	1	1	7	1	3	7	5	Mild
102	28	M	1	2	120			No	No	No	No	No	Yes	No		13.1	101	0.82	AR, Spur to R	AR	AR	0	1	0	0	0	0	1	0	2	1	0	2	1	Anosmia
103	39	M	2	2	4		DM	Yes	No	No	No	No	Yes	No		15	194/213	0.81	DNS to R, AR	AR	AR	0	1	1	1	1	1	1	1	7	1	0	7	3.5	Severe
104	53	F	1	2	12			No	No	No	No	No	No	No		12.7	118	0.59	Normal	Anosmia	Idopathic	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
105	33	M	1	2	84			No	No	No	No	No	No	No		17	53/84	0.96	DNS to L	Anosmia	Idopathic	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
106	36	M	1	2	60		SHTN	No	Yes	No	No	No	No	Yes		15.2	123	1.27	CRS with NP	CRS with NP	CRS with NP	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
107	55	F	1	2	84			No	No	No	No	No	No	No		13.2	99/148		Normal	LPR / Anosmia	Idopathic	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
108	45	M	1	2	60			No	No	No	No	No	No	Yes	Right VC keratosis	14.8	90	1.04	CRS with NP	CRS with NP	CRS with NP	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
109	58	M	1	1	7		B/L CSOM	No	No	No	No	No	No	No	RTA preceding Anosmia	14.4	103	0.95	DNS to R	Traumatic Anosmia	Post traumatic Anosmia	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
110	28	F	1	2	3			No	No	No	No	No	No	No		13.9	93	0.64	DNS to L	Anosmia	Idopathic	0	0	0	0	0	0	0	0	0	1	0	0	0	Anosmia
111	24	M	3	2	6			No	No	No	No	No	Yes	No		15.3	103	0.99	AR	AR, Anosmia	AR	1	1	1	1	1	1	1	1	7	1	1	7	4	Moderate
112	38	M	3	2	3		SHTN	No	Yes	No	No	No	No	No		11.2	108	0.86	DNS to L, B/L ETD	B/L ETD, Anosmia	Idopathic	2	1	1	1	1	0	1	1	6	1	2	6	4	Moderate
113	44	F	2	2	24			No	No	No	No	No	Yes	No		9.5	155	0.65	AR, B/L ETD	AR, B/L ETD	AR	0	1	1	1	0	0	0	1	4	1	0	4	2	Severe
114	55	F	1	2	72		DM, SHTN	Yes	Yes	No	No	No	Yes	No		10.9	126	0.85	AR	AR, Anosmia	AR	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
115	50	F	0	0				No	No	No	No	No	No	No							Idopathic	6	1	1	1	1	1	1	1	7	1	6	7	6.5	Normosmia
116	32	M	2	0				No	No	No	No	No	Yes	No		14.5	99	1.06	AR	AR	AR	3	1	0	1	0	0	1	1	4	1	3	4	3.5	Severe
117	48	M	1	0				No	No	No	No	No	No	No		12.1	85	0.68	DNS to L, spur to R	AR, LPR, Left maxillary CRS	AR	1	0	0	1	0	0	0	1	2	1	1	2	1.5	Anosmia
118	55	M	1	0				No	No	No	No	No	No	Yes	Post op FESS	14.9	122	0.62	B/L CRS with NP	B/L CRS with NP	CRS with NP	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
119	62	M	1	2	48			No	No	No	No	No	No	No		14	107	0.93	High DNS to R	Anosmia	Idopathic	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
120	57	F	1	2	6	URTI	Hypothyroid, Dyslipidemia	No	No	Yes	No	Yes	No	No		13.4	107	0.86	MildDNS to R	Sinusitis, Anosmia	CRS without NP	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
121	36	F	2	0				No	No	No	No	No	No	No	Pansinusitis	13.6	86	0.7	DNS to L	Pansinusitis, Anosmia	CRS without NP	0	1	1	1	1	1	1	1	7	1	0	7	3.5	Severe
122	62	M	1	2	36		SHTN	No	Yes	No	No	No	No	No		12.7	99	0.82	High DNS to L	Anosmia	Idopathic	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
123	18	M	2	0				No	No	No	No	No	No	No		14.2	88	0.94	DNS with spur to L	AR, Anosmia	AR	1	1	1	1	1	0	1	1	6	1	1	6	3.5	Severe

Sl.No.	Age	Sex	Group	Onset of loss of smell	Duration (Months)	History of prior viral illness	Comorbidities	Diabetes	Hypertension	Hypothyroidism	Bronchial asthma	Dyslipidemia	AR	CRS	Others	Hb	RBS	Creatinine	RNE	Diagnosis	Nasal Disease	Test Olfactory threshold	Odour identification test: Cinnamon	Asafoetida	Coffee	Tea	Pepper	Clove oil	Baby powder	Total correct	Eucalyptus (Trigeminal)	Score: Olfactory threshold	Odour identification	Composite score	Types of olfactory dysfunction
124	34	M	4	0				No	No	No	No	No	Yes	No		13.7	92	1.12	AR	AR	AR	3	1	1	1	1	1	1	1	7	1	3	7	5	Mild
125	48	F	1	2	12			No	No	No	No	No	No	No		13.2	89	0.65	Spur to L	Anosmia	Idopathic	0	0	0	0	0	0	0	0	0	1	0	0	0	Anosmia
126	45	F	3	2	24		DM	Yes	No	No	No	No	Yes	No		10.3	139/222		AR, DNS to L	AR, Anosmia	AR	2	1	1	1	1	1	1	0	6	1	2	6	4	Moderate
127	37	F	2	2	120			No	No	No	No	No	Yes	No		12.3	82	0.88	AR, DNS with spur to R	AR, Anosmia	AR	1	1	1	1	1	0	1	1	6	1	1	6	3.5	Severe
128	15	F	1	2	84			No	No	No	No	No	No	No					Normal	Anosmia	Idopathic	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
129	36	F	1	2	120			No	No	No	No	No	No	No		13.5		0.6	Atrophic rhinitis	Atrophic rhinitis	Atrophic rhinitis	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
130	49	M	1	2	240			No	No	No	No	No	No	No		16.3	91	1.09	High DNS to L, spur to R	Anosmia	Idopathic	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
131	57	M	2	1	3	Fever	DM	Yes	No	No	No	No	No	No		10.9	287/355	0.86	Normal	Anosmia	Idopathic	2	0	0	1	1	1	1	1	5	1	2	5	3.5	Severe
132	40	M	2	2	120		DM, SHTN	Yes	Yes	No	No	No	No	Yes					CRS with NP	CRS with NP	CRS with NP	1	1	1	1	1	0	1	0	5	1	1	5	3	Severe
133	29	M	4	2	120		SHTN	No	Yes	No	No	No	Yes	No		15.4	86	0.84	AR, DNS to R	AR, Anosmia	AR	3	1	1	1	1	1	1	1	7	1	3	7	5	Mild
134	51	F	1	2	8		SHTN	No	Yes	No	No	No	No	No		12.6	132	0.67	LPR, ETD	LPR, ETD	Idopathic	0	0	0	0	0	0	0	0	0	1	0	0	0	Anosmia
135	49	F	1	2	72			No	No	No	No	No	No	No		12.9	99	0.51	Normal	Anosmia	Idopathic	0	0	0	0	0	0	0	0	0	1	0	0	0	Anosmia
136	40	F	2	0				No	No	No	No	No	Yes	No		13.5	91	0.62	AR, B/L ETD, DNS with spur	AR, B/L ETD	AR	0	1	1	0	0	1	1	0	4	1	0	4	2	Severe
137	36	M	3	0			Hypothyroid	No	No	Yes	No	No	Yes	No		18.6	77	1	AR, spur to R	AR, Anosmia	AR	2	1	1	1	1	1	1	0	6	1	2	6	4	Moderate
138	53	F	2	2	24		Hypothyroid, SHTN, CAD, BA	No	Yes	Yes	Yes	No	No	Yes		12.6	122	0.83	CRS with NP	CRS with NP	CRS with NP	0	1	1	1	1	1	1	0	6	1	0	6	3	Severe
139	55	F	2	1	12	Fever	DM, SHTN	Yes	Yes	No	No	No	Yes	No	Parosmia	11.8	153/244	0.48	AR	AR, Anosmia	AR	0	1	1	1	0	0	1	1	5	1	0	5	2.5	Severe
140	21	M	3	2	48			No	No	No	No	No	Yes	No		16.2	99	1.13	AR	AR, Anosmia	AR	1	1	1	1	1	1	1	1	7	1	1	7	4	Moderate
141	40	F	1	2				No	No	No	No	No	Yes	No		13.1	95	0.65	AR	AR, Anosmia	AR	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
142	48	F	4	0			BA	No	No	No	Yes	No	No	Yes		12.9	93	0.7	CRS with NP	CRS with NP	CRS with NP	3	1	1	1	1	1	1	1	7	1	3	7	5	Mild
143	44	M	1	2	30			No	No	No	No	No	No	No	Post op septoplasty and FESS	14.6	91	0.93	Grade 2 SNP left , multiple synechiae	Grade 2 SNP left nose	SNP	0	0	0	0	0	0	0	0	0	1	0	0	0	Anosmia
144	56	M	1	2	36		Dyslipidemia	No	No	No	No	Yes	No	No	Post op R hemithyroidectomy	14	156	1.39	normal	Anosmia	Idopathic	0	0	0	0	0	0	0	0	0	1	0	0	0	Anosmia
145	23	M	3	0				No	No	No	No	No	No	Yes		14.1	94	1.19	CRS with NP	CRS with NP	CRS with NP	1	1	1	1	1	1	1	1	7	1	1	7	4	Moderate
146	30	M	1	2	12			No	No	No	No	No	Yes	No		14	89	0.82	AR	AR	AR	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
147	48	F	1	0			DM, BA	Yes	No	No	Yes	No	No	Yes		13	317/456	0.61	CRS with NP	CRS with NP	CRS with NP	0	0	0	0	0	0	0	0	0	1	0	0	0	Anosmia
148	50	F	1	2	12		SHTN	No	Yes	No	No	No	No	No		12.1	64/85	0.93	DNS with spur to R	Anosmia	Idopathic	0	0	0	0	0	0	0	0	0	1	0	0	0	Anosmia
149	35	F	3	2	2		Hypothyroid	No	No	Yes	No	No	No	Yes		13.1	94	0.82	CRS with NP	CRS with NP	CRS with NP	2	1	1	1	1	1	1	1	7	1	2	7	4.5	Moderate
150	53	M	1	2	120		BA	No	No	No	Yes	No	Yes	No					AR, DNS to R	AR, Anosmia	AR	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
151	63	F	1	1	48	RTA	DM	Yes	No	No	No	No	Yes	No		11.9	43/65	0.65	AR, Left sphenoid sinusitis	AR, Sinusitis	CRS without NP	0	1	0	1	0	1	0	0	3	1	0	3	1.5	Anosmia
152	27	M	2	1	8	Fall		No	No	No	No	No	No	No		15	87	0.79	Bilateral DNS with spur to R	Anosmia	Idopathic	0	1	0	1	1	0	0	1	4	1	0	4	2	Severe
153	43	F	1	2	48			No	No	No	No	No	Yes	No		13.5	105	0.58	AR, DNS with spur to R	AR, Anosmia	AR	0	0	0	0	0	0	0	0	0	1	0	0	0	Anosmia
154	38	M	2	2	2			No	No	No	No	No	No	No					DNS to R	Anosmia	Idopathic	0	1	1	0	1	0	1	1	5	1	0	5	2.5	Severe
155	41	M	1	2	12			No	No	No	No	No	No	Yes		14.5	83	0.82	CRS with NP	CRS with NP	CRS with NP	0	0	0	0	0	0	0	0	0	1	0	0	0	Anosmia
156	36	M	1	1	1	RTA		No	No	No	No	No	Yes	No		18.5		0.88	AR, DNS to L	AR, Anosmia	AR	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
157	44	M	1	2	24		IGT	No	No	No	No	No	No	Yes		12.2	121/170	0.84	CRS with NP, DNS to L	CRS with NP	CRS with NP	0	0	0	0	0	0	0	0	0	1	0	0	0	Anosmia
158	54	F	1	2	36		SHTN	No	Yes	No	No	No	Yes	No				O.97	AR	AR, Anosmia	AR	0	0	0	0	0	0	0	0	0	1	0	0	0	Anosmia
159	43	F	1	2	72		Hypothyroid	No	No	Yes	No	No	Yes	No		11.1	102	0.56	AR, B/L ETD	AR, B/L ETD	AR	0	1	1	0	0	0	1	0	3	1	0	3	1.5	Anosmia
160	36	F	2	2	36			No	No	No	No	No	No	Yes		12.4	98	0.51	CRS, DNS to L	CRS	CRS without NP	1	1	1	1	1	0	1	0	5	1	1	5	3	Severe
161	61	F	1	1	6	Brain aneurysm surgery	SHTN	No	Yes	No	No	No	No	No		12	134	0.97	DNS with spur to R	Post op left ICA aneurysm Anosmia	Idopathic	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
162	33	F	4	2	6			No	No	No	No	No	Yes	No		12.5	98	0.6	AR	AR	AR	3	1	1	1	1	1	1	1	7	1	3	7	5	Mild
163	40	F	1	0				No	No	No	No	No	No	No		12.9	130	0.89	Spur to L	AR	AR	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
164	35	M	1	2	180			No	No	No	No	No	No	Yes		12.8	79		DNS to R, spur to L, CRS	CRS	CRS without NP	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
165	18	M	1	1	2	RTA		No	No	No	No	No	No	No		12.2		0.59	DNS to L	Post traumatic Anosmia	Post traumatic Anosmia	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
166	52	M	1	2	60			No	No	No	No	No	No	Yes		15.4	133	0.91	CRS with NP	CRS with NP, AR	CRS with NP	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
167	36	M	2	2	240		DM, SHTN	Yes	Yes	No	No	No	Yes	No		12.2	113	0.82	AR	AR	AR	3	1	0	1	0	0	1	0	3	1	3	3	3	Severe
168	28	M	2	0				No	No	No	No	No	No	No		17.6	107	0.89	Right nasal mass	REAH/ SNP	SNP	0	1	1	1	1	1	1	1	7	1	0	7	3.5	Severe
169	34	F	1	2	2		BA	No	No	No	Yes	No	Yes	No		11.5		0.56	AR	AR	AR	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
170	40	F	1	2	36		DM	Yes	No	No	No	No	No	No		13.9	217		High DNS to L	Anosmia	Idopathic	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
171	36	F	2	2	360			No	No	No	No	No	No	No		13.1	104		DNS to R, septal spur	Anosmia	Idopathic	0	1	1	1	1	0	1	0	5	1	0	5	2.5	Severe
172	27	F	3	0				No	No	No	No	No	No	No		12.1	94	0.68	DNS to L with septal spur	Vasomotor rhinopathy	Idopathic	3	1	1	1	1	0	1	0	5	1	3	5	4	Moderate
173	27	M	2	0				No	No	No	No	No	No	Yes		15.8	101	0.92	CRS	CRS, AR	CRS without NP	0	1	1	1	0	0	1	0	4	1	0	4	2	Severe
174	21	M	2	0				No	No	No	No	No	No	Yes		13.2	95	1.01	CRS	CRS	CRS without NP	0	1	1	1	1	1	1	1	7	1	0	7	3.5	Severe
175	29	M	2	0				No	No	No	No	No	No	No		14.3	106	0.87	DNS to L with septal spur	AR	AR	0	1	1	1	1	0	1	0	5	1	0	5	2.5	Severe
176	46	M	2	1	2	Fever		No	No	No	No	No	No	No		14.4	258	0.97	DNS to L with spur	Anosmia	Idopathic	0	1	1	1	0	0	1	1	5	1	0	5	2.5	Severe
177	52	F	2	2	240		DM	Yes	No	No	No	No	No	No	LPRD	11.7	331/411	0.77	High DNS to R	Anosmia	Idopathic	0	1	1	1	1	1	1	1	7	1	0	7	3.5	Severe
178	37	M	3	0				No	No	No	No	No	No	Yes		11.8	106	0.79	CRS with NP	AFRS with Pansinusitis, AR	CRS with NP	1	1	1	1	1	1	1	1	7	1	1	7	4	Moderate
179	59	M	4	0			SHTN, BA	No	Yes	No	Yes	No	Yes	No		14.9	62/103	0.91	AR, Bilateral SNP	AR, Bilateral SNP	SNP	3	1	1	1	1	1	1	1	7	1	3	7	5	Mild
180	37	M	2	0				No	No	No	No	No	Yes	No		15	126	0.92	AR, DNS with spur to R	AR, Rhinitis medicamentosa	AR	0	1	1	1	1	1	1	1	7	1	0	7	3.5	Severe
181	35	F	4	2	4			No	No	No	No	No	Yes	No		13.5			AR, DNS with spur to L	AR	AR	3	1	1	1	1	1	1	1	7	1	3	7	5	Mild
182	44	M	4	2	12		DM	Yes	No	No	No	No	Yes	No		15.8	101/106	1	AR	AR	AR	3	1	1	1	1	1	1	1	7	1	3	7	5	Mild
183	48	M	1	0			SHTN	No	Yes	No	No	No	Yes	No		16.7	93	0.92	AR, DNS	AR	AR	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
184	29	F	3	0			DM, SHTN	Yes	Yes	No	No	No	No	No		12	116	0.91	DNS to R, spur to L, R ETD	AR	AR	2	1	1	1	1	1	1	1	7	1	2	7	4.5	Moderate
185	60	M	2	2	24		IHD	No	No	No	No	No	No	No		13.3	111	1.1	Normal	Anosmia	Idopathic	1	1	0	1	1	1	1	1	6	1	1	6	3.5	Severe
186	40	M	3	2	240			No	No	No	No	No	Yes	No		15	94	0.91	AR, DNS with spur to R	AR	AR	4	0	0	1	1	1	1	1	5	1	4	5	4.5	Moderate
187	63	F	1	1	6			No	No	No	No	No	Yes	No		12.4	95	0.71	AR	AR	AR	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
188	34	M	0	0				No	No	No	No	No	No	No		14.4	121	0.92	DNS with spur to R	Anosmia	Idopathic	5	1	1	1	1	1	1	1	7	1	5	7	6	Normosmia
189	22	M	3	0				No	No	No	No	No	No	No		15.8	100	1.01	Bilateral high DNS	AR	AR	2	1	1	1	1	1	1	1	7	1	2	7	4.5	Moderate
190	43	M	1	1	1	Fever	SHTN	No	Yes	No	No	No	No	No		13.3	88	0.84	DNS to L with spur	Anosmia	Idopathic	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
191	30	F	0	0				No	No	No	No	No	No	No		13.4	84	0.71	Normal	AR	AR	6	1	1	1	1	1	1	1	7	1	6	7	6.5	Normosmia
192	24	M	1	1	5	RTA		No	No	No	No	No	No	No		14.8	76	0.72	DNS to R	Post traumatic Anosmia	Post traumatic Anosmia	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
193	34	M	4	2	264		BA	No	No	No	Yes	No	No	No	Multiple nasal surgeries	13.7	97	1.04	Post op status - FESS	CRS Post op	Post FESS	4	1	1	1	1	1	1	1	7	1	4	7	5.5	Mild
194	46	M	4	0			DM	Yes	No	No	No	No	No	No		14.4	207	0.62	DNS to R	CRS with NP	CRS with NP	3	1	1	1	1	1	1	1	7	1	3	7	5	Mild
195	47	M	1	2	120		DM	Yes	No	No	No	No	Yes	No		16.6	122	0.85	AR, B/L ETD	AR	AR	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
196	64	M	2	2	72			No	No	No	No	No	No	Yes	Bilateral FESS	13.9	96	1.11	CRS, DNS to R	CRS, AR	CRS without NP	0	1	1	1	1	0	1	1	6	1	0	6	3	Severe
197	42	M	2	2	18			No	No	No	No	No	No	No		15.6		0.95	Normal	Anosmia	Idopathic	2	1	1	1	1	0	1	0	5	1	2	5	3.5	Severe
198	17	M	3	0			BA	No	No	No	Yes	No	Yes	No		14.9	77	0.88	AR, DNS with spur to R	AR	AR	2	1	1	1	1	0	1	1	6	1	2	6	4	Moderate
199	54	M	1	2	12		SHTN	No	Yes	No	No	No	Yes	No		14.8	111	1.2	AR	AR, Anosmia	AR	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
200	52	M	2	0				No	No	No	No	No	Yes	No		15	135	1.19	AR, DNS with spur to L	AR	AR	0	1	1	1	1	1	1	1	7	1	0	7	3.5	Severe
201	34	M	1	2	24			No	No	No	No	No	No	No		15.9	90	0.97	Atrophic rhinitis	Atrophic rhinitis, Anosmia	Atrophic rhinitis	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
202	21	M	1	2	48			No	No	No	No	No	No	No		15	109	0.85	Atrophic rhinitis	Atrophic rhinitis, Anosmia	Atrophic rhinitis	3	0	0	0	0	0	0	0	0	0	3	0	1.5	Anosmia
203	25	M	3	1	252		DM	Yes	No	No	No	No	Yes	No		16.6	312	0.91	AR	Post traumatic Anosmia	Post traumatic Anosmia	2	1	1	1	1	1	1	1	7	1	2	7	4.5	Moderate
204	24	M	2	0				No	No	No	No	No	No	No		15.5	83	0.83			Idopathic	0	1	1	1	1	0	1	0	5	1	0	5	2.5	Severe
205	58	M	1	0				No	No	No	No	No	No	No		15.7	119	0.79	Atrophic rhinitis	Atrophic rhinitis	Atrophic rhinitis	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
206	26	M	1	2	48			No	No	No	No	No	No	No		16.1	88	1.15	Atrophic rhinitis	Atrophic rhinitis, Anosmia	Atrophic rhinitis	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
207	51	M	0	2	12			No	No	No	No	No	No	No		14.2	110	0.89	High DNS to B/L	Anosmia	Idopathic	6	1	1	1	1	1	1	1	7	1	6	7	6.5	Normosmia
208	72	M	1	2	6			No	No	No	No	No	No	No					DNS to L	Anosmia	Idopathic	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
209	43	M	4	2	108		SHTN	No	Yes	No	No	No	Yes	No				0.98	AR	AR, Anosmia	AR	4	1	1	1	1	1	1	1	7	1	4	7	5.5	Mild
210	43	F	1	2	12			No	No	No	No	No	No	Yes					CRS	CRS	CRS without NP	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
211	46	M	1	2	180			No	No	No	No	No	No	Yes		13.2	96	0.9	CRS	CRS	CRS without NP	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
212	59	M	1	2	300			No	No	No	No	No	Yes	No		12.6	153	1.08	AR	AR, Anosmia	AR	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
213	52	F	4	2	48		BA	No	No	No	Yes	No	Yes	No		12.3	101		AR	AR, Anosmia	AR	4	1	1	1	1	1	1	1	7	1	4	7	5.5	Mild
214	71	F	1	2	36			No	No	No	No	No	Yes	No		11.7	86	0.57	AR	AR, Anosmia	AR	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
215	33	F	1	2	60			No	No	No	No	No	No	No		12.9	126	0.59	Atrophic rhinitis	Atrophic rhinitis	Atrophic rhinitis	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
216	61	M	3	2	7			No	No	No	No	No	Yes	No					AR	AR	AR	4	1	0	1	1	0	1	0	4	1	4	4	4	Moderate
217	24	M	4	0				No	No	No	No	No	No	No		14.6	88	0.99	DNS with spur to L	AR, B/L maxillary sinusitis	AR	3	1	1	1	1	1	1	1	7	1	3	7	5	Mild
218	40	F	1	2	4			No	No	No	No	No	No	No		8.8	90	0.66	DNS to L	AR	AR	0	0	0	1	0	0	0	0	1	1	0	1	0.5	Anosmia
219	65	M	1	2	12		DM, SHTN, COPD	Yes	Yes	No	No	No	Yes	No		13.3	117	1.37	AR, B/L ETD	AR, Anosmia	AR	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
220	55	F	1	2	72		SHTN	No	Yes	No	No	No	Yes	No		12.1	327	0.81	AR	AR, Anosmia	AR	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
221	41	M	1	1	36	RTA		No	No	No	No	No	Yes	No		14.8	108	1.08	AR	Post traumatic Anosmia	Post traumatic Anosmia	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
222	30	M	2	2	24			No	No	No	No	No	Yes	No		14.1	85	0.7	AR	AR, Anosmia	AR	0	0	0	1	1	1	1	0	4	1	0	4	2	Severe
223	31	F	1	1	1	URTI, Fever		No	No	No	No	No	Yes	No		14			AR	Post viral Anosmia	Post viral Anosmia	0	1	1	1	0	0	0	0	3	1	0	3	1.5	Anosmia
224	38	M	1	2	120		Hepatitis B	No	No	No	No	No	Yes	No		16.2		1	AR, B/L ETD, Spur to L	AR, Anosmia, Hep B	AR	0	0	1	0	0	0	0	0	1	1	0	1	0.5	Anosmia
225	37	M	3	2	48		BA	No	No	No	Yes	No	No	No		13.5	95	0.95	DNS with spur to L	AR	AR	2	1	1	1	1	1	1	1	7	1	2	7	4.5	Moderate
226	35	F	1	0				No	No	No	No	No	No	Yes		12.2	78	0.62	DNS with spur to R, CRS with NP	CRS with NP	CRS with NP	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
227	39	M	3	0			SHTN	No	Yes	No	No	No	Yes	No		12.9	105	1.12	AR, DNS to L	AR	AR	2	1	1	1	1	1	1	1	7	1	2	7	4.5	Moderate
228	44	M	1	2	120			No	No	No	No	No	No	No	Post op B/L Youngs	16.4	92		Post op status - Youngs	Atrophic rhinitis	Atrophic rhinitis	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
229	57	F	4	0			SHTN	No	Yes	No	No	No	No	No		11.5		0.61	DNS with spur to L	Vasomotor rhinitis	Idopathic	5	0	1	1	0	1	1	1	5	1	5	5	5	Mild
230	61	M	1	2	240			No	No	No	No	No	No	Yes		14.8	100	0.97	CRS with NP	CRS with NP	CRS with NP	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
231	48	M	2	2	24			No	No	No	No	No	No	No		13.2	83	0.84	DNS with spur to R	Anosmia	Idopathic	0	1	1	1	1	1	1	0	6	1	0	6	3	Severe
232	32	F	1	1	3	RTA		No	No	No	No	No	Yes	No		11.5		0.55	AR, DNS to R	Post traumatic Anosmia	Post traumatic Anosmia	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
233	37	F	3	0				No	No	No	No	No	No	Yes		12.9	113	0.57	CRS with NP	CRS with NP	CRS with NP	2	1	1	1	1	1	1	1	7	1	2	7	4.5	Moderate
234	34	M	3	0				No	No	No	No	No	No	No							Idopathic	2	1	1	1	1	1	1	1	7	1	2	7	4.5	Moderate
235	41	F	4	0				No	No	No	No	No	Yes	No		14	87		AR	AR	AR	3	1	1	1	1	1	1	1	7	1	3	7	5	Mild
236	47	F	2	0			Hypothyroid	No	No	Yes	No	No	No	Yes		10.2	91	0.74	CRS with NP, DNS to L	CRS with NP	CRS with NP	0	1	1	1	1	0	1	1	6	1	0	6	3	Severe
237	51	M	1	1	18	Fall		No	No	No	No	No	Yes	No		12.7		0.89	AR	Post traumatic Anosmia	Post traumatic Anosmia	0	0	0	0	0	0	0	1	1	1	0	1	0.5	Anosmia
238	14	F	3	0				No	No	No	No	No	No	No	Right frontoethmoidal mucocele	12.4	100	0.67	Right mucocele	Right mucocele	Post FESS	1	1	1	1	1	1	1	1	7	1	1	7	4	Moderate
239	33	M	2	2	3			No	No	No	No	No	No	No					DNS to R	Anosmia	Idopathic	0	1	1	1	1	1	0	1	6	1	0	6	3	Severe
240	36	M	1	2	240			No	No	No	No	No	No	Yes		15.2	111	1.05	CRS with NP	CRS with NP	CRS with NP	1	0	0	0	1	0	0	1	2	1	1	2	1.5	Anosmia
241	43	M	4	0			Hypothyroid	No	No	Yes	No	No	No	Yes		14.4	123	0.84	CRS with NP	CRS with NP	CRS with NP	3	1	1	1	1	1	1	1	7	1	3	7	5	Mild
242	47	F	2	2	12			No	No	No	No	No	No	No		12	90	0.64	DNS with spur to L	Anosmia	Idopathic	0	1	0	1	1	0	1	0	4	1	0	4	2	Severe
243	42	M	3	2	60			No	No	No	No	No	No	Yes		16.1	82	1.22	CRS with NP	CRS with NP	CRS with NP	1	1	1	1	1	1	1	1	7	1	1	7	4	Moderate
244	40	F	1	1	1	Fever		No	No	No	No	No	No	No		13.7	206	0.76	Normal	Post viral Anosmia	Post viral Anosmia	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
245	44	M	2	0				No	No	No	No	No	Yes	No		12.4	119	0.78	AR	AR	AR	0	1	1	1	1	1	1	1	7	1	0	7	3.5	Severe

Sl.No.	Age	Sex	Group	Onset of loss of smell	Duration (Months)	History of prior viral illness	Comorbidities	Diabetes	Hypertension	Hypothyroidism	Bronchial asthma	Dyslipidemia	AR	CRS	Others	Hb	RBS	Creatinine	RNE	Diagnosis	Nasal Disease	Test Olfactory threshold	Odour identification test Cinnamon	Asafoetida	Coffee	Tea	Pepper	Clove oil	Baby powder	Total correct	Eucalyptus (Trigeminal)	Score Olfactory threshold	Odour identification	Composite score	Types of olfactory dysfunction
246	31	F	3	2	48			No	No	No	No	No	Yes	No					AR, DNS to L with spur to R	AR, Anosmia	AR	2	1	1	1	1	1	1	1	7	1	2	7	4.5	Moderate
247	46	M	3	2	4			No	No	No	No	No	No	No		15	101	0.96	Sharp spur to R	Anosmia	Idopathic	2	1	1	1	1	1	1	1	7	1	2	7	4.5	Moderate
248	42	F	4	2	240		Hypothyroid	No	No	Yes	No	No	Yes	No			118	0.73	AR	AR, Anosmia	AR	3	1	1	1	1	1	1	1	7	1	3	7	5	Mild
249	53	F	1	2	120		SHTN	No	Yes	No	No	No	No	Yes		13	129	0.66	CRS with fungal ball L	CRS	CRS wiyhout NP	0	0	0	0	0	0	0	0	0	1	0	0	0	Anosmia
250	31	F	3	2	36			No	No	No	No	No	Yes	No		12.7	129	0.76	AR, DNS with spur to L	AR, Anosmia	AR	1	1	1	1	1	1	1	1	7	1	1	7	4	Moderate
251	52	M	3	0			SHTN	No	Yes	No	No	No	No	No		14.1	118	0.87	R SNP	SNP	SNP	1	1	1	1	1	1	1	1	7	1	1	7	4	Moderate
252	23	M	3	0				No	No	No	No	No	Yes	No		15.3	118	0.88	AR, DNS with spur to R	AR	AR	1	1	1	1	1	1	1	1	7	1	1	7	4	Moderate
253	33	F	3	0				No	No	No	No	No	Yes	No		13	80	0.6	AR, DNS to R	AR	AR	2	1	1	1	1	1	1	1	7	1	2	7	4.5	Moderate
254	25	M	2	2	5			No	No	No	No	No	Yes	No		15.6	89	0.96	AR	AR, Anosmia	AR	0	1	1	1	1	0	1	1	6	1	0	6	3	Severe
255	37	M	1	0				No	No	No	No	No	Yes	No		15.5	87		AR, B/L ETD, DNS to R, POST OP	AR	AR	1	0	0	1	0	0	0	0	1	1	1	1	1	Anosmia
256	33	M	2	0			SHTN	No	Yes	No	No	No	Yes	No		14.9	396	0.87	AR, DNS with spur to R	AR	AR	3	1	1	1	1	0	0	0	4	1	3	4	3.5	Severe
257	22	M	3	0				No	No	No	No	No	Yes	No		15.9	86	0.88	AR, DNS to R with spur to L	AR	AR	4	1	1	1	0	1	0	1	5	1	4	5	4.5	Moderate
258	36	M	2	2	84			No	No	No	No	No	No	No		13.8	104	1.04	Atrophic rhinitis	Atrophic rhinitis	Atrophic rhinitis	0	1	0	1	1	1	1	1	6	1	0	6	3	Severe
259	50	F	3	0			Hypothyroid	No	No	Yes	No	No	Yes	No		11.9	159	0.73	AR	AR	AR	2	1	1	1	1	1	1	1	7	1	2	7	4.5	Moderate
260	21	F	4	0				No	No	No	No	No	Yes	No		10.6	96	0.48	AR	AR, Anosmia	AR	3	1	1	1	1	1	1	1	7	1	3	7	5	Mild
261	23	M	3	2	36			No	No	No	No	No	Yes	No		16.2	84	0.77	AR	AR, Anosmia	AR	2	1	1	1	1	0	1	1	6	1	2	6	4	Moderate
262	30	F	3	2	36			No	No	No	No	No	Yes	No		11.6	89	0.61	AR, DNS to L	AR, Anosmia	AR	2	1	1	1	1	1	1	1	7	1	2	7	4.5	Moderate
263	33	M	1	1	144	RTA		No	No	No	No	No	No	No		13.9	93	0.8	SNP	Post traumatic Anosmia	Post traumatic Anosmia	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
264	22	M	3	0				No	No	No	No	No	Yes	No		15.2	88	0.79	AR	AR	AR	2	1	1	1	1	1	1	1	7	1	2	7	4.5	Moderate
265	42	M	1	1	30	RTA		No	No	No	No	No	No	No					High DNS to R	Traumatic Anosmia	Post traumatic Anosmia	0	1	1	0	0	0	0	0	2	1	0	2	1	Anosmia
266	33	F	2	2	30		BA	No	No	No	Yes	No	Yes	No		13.1	104	0.64	AR	AR, Anosmia	AR	0	1	1	1	1	1	1	1	7	1	0	7	3.5	Severe
267	50	F	1	2	72			No	No	No	No	No	No	No		14.6	94	0.72	Spur to L	Anosmia	Idopathic	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
268	36	M	4	2	180			No	No	No	No	No	Yes	No		15.3	103	0.79	AR, DNS to L with spur, R concha	AR, Anosmia	AR	3	1	1	1	1	1	1	1	7	1	3	7	5	Mild
269	36	M	4	0			SHTN	No	Yes	No	No	No	No	Yes		15.4	133	0.95	CRS with NP	CRS with NP	CRS with NP	3	1	1	1	1	1	1	1	7	1	3	7	5	Mild
270	26	M	3	0				No	No	No	No	No	Yes	Yes		12.8	124	0.89	AR, CRS	AR, CRS	AR	3	1	1	1	1	0	0	1	5	1	3	5	4	Moderate
271	58	F	4	0				No	No	No	No	No	Yes	No		12.7	89	0.6	AR	AR	AR	3	1	1	1	1	1	1	1	7	1	3	7	5	Mild
272	47	F	4	0				No	No	No	No	No	Yes	No		11.6	80	0.66	AR	AR	AR	3	1	1	1	1	1	1	1	7	1	3	7	5	Mild
273	31	F	1	2	48			No	No	No	No	No	No	Yes		13.5	90	0.6	CRS with NP	CRS with NP	CRS with NP	0	0	0	0	0	0	0	0	0	1	0	0	0	Anosmia
274	45	M	3	0			SHTN, Dyslipidemia	No	Yes	No	No	Yes	Yes	No		15.1	163	0.74	AR, DNS to L	AR	AR	3	1	1	1	1	1	1	0	6	1	3	6	4.5	Moderate
275	38	M	1	1	24	RTA		No	No	No	No	No	No	No					DNS to R	Traumatic Anosmia	Post traumatic Anosmia	0	0	0	0	0	0	0	0	0	1	0	0	0	Anosmia
276	30	M	3	0				No	No	No	No	No	Yes	No		14.9	100	0.82	R SNP, AR	SNP, AR	SNP	4	1	1	1	1	1	0	0	5	1	4	5	4.5	Moderate
277	20	M	3	0				No	No	No	No	No	Yes	No		15.2	88	0.72	High DNS to R, AR	AR	AR	4	1	1	1	1	0	1	0	5	1	4	5	4.5	Moderate
278	42	M	3	0				No	No	No	No	No	Yes	No		15	110	0.92	AR, DNS to R	AR	AR	2	1	1	1	1	1	1	1	7	1	2	7	4.5	Moderate
279	38	F	2	0				No	No	No	No	No	Yes	No		13.4	92	0.62	DNS to R, AR	AR	AR	2	1	1	1	1	0	0	0	4	1	2	4	3	Severe
280	47	M	1	2	120			No	No	No	No	No	No	No					Post op status - FESS	CRS with NP	CRS with NP	0	0	0	0	0	0	0	0	0	1	0	0	0	Anosmia
281	47	M	2	2	72			No	No	No	No	No	No	No		14	102	0.95	DNS to R	Anosmia	Idopathic	0	1	1	1	1	0	1	1	6	1	0	6	3	Severe
282	32	F	2	0				No	No	No	No	No	No	No		11	104	0.55	B/L ITH	AR	AR	1	1	1	1	1	0	1	1	6	1	1	6	3.5	Severe
283	41	F	1	0				No	No	No	No	No	No	Yes		13.2		0.48	CRS with NP	CRS with NP	CRS with NP	0	1	1	0	0	0	0	0	2	1	0	2	1	Anosmia
284	30	M	3	0				No	No	No	No	No	Yes	No		13.5	90	0.86	AR, DNS to L	AR	AR	2	1	1	1	1	0	1	1	6	1	2	6	4	Moderate
285	55	F	1	0				No	No	No	No	No	Yes	No		10.3	89	0.48	AR	AR	AR	0	0	1	0	0	0	0	0	1	1	0	1	0.5	Anosmia
286	37	M	4	0				No	No	No	No	No	Yes	No		13.3	93	1.14	AR, DNS to L with spur to L	AR	AR	3	1	1	1	1	1	1	1	7	1	3	7	5	Mild
287	34	M	3	2	84		Dyslipidemia	No	No	No	No	Yes	Yes	No		13.6	110	0.77	AR, DNS with spur to L	AR, Anosmia	AR	2	1	1	1	1	1	1	1	7	1	2	7	4.5	Moderate
288	33	F	2	0			Hypothyroid	No	No	Yes	No	No	No	No		12.4	110	0.62	Gross DNS to R	Anosmia	Idopathic	2	1	1	1	1	0	1	0	5	1	2	5	3.5	Severe
289	32	M	1	0				No	No	No	No	No	No	No	Post op FESS	15.6	94	0.86	B/L SNP, septal perforation	Recurrent SNP	SNP	0	0	0	0	0	0	0	0	0	1	0	0	0	Anosmia
290	44	M	4	2	96			No	No	No	No	No	No	Yes		13.9	94	0.86	CRS without NP, spur to L	CRS without NP	CRS without NP	3	1	1	1	1	1	1	1	7	1	3	7	5	Mild
291	39	M	1	2	240			No	No	No	No	No	No	No		12.2	92	0.69	Atrophic rhinitis	Atrophic rhinitis, Anosmia	Atrophic rhinitis	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
292	38	M	1	2	36			No	No	No	No	No	Yes	No		13.9	102	1.06	AR, B/L ETD, DNS with spur	AR, Anosmia	AR	1	1	1	0	0	0	0	0	2	1	1	2	1.5	Anosmia
293	49	F	3	0				No	No	No	No	No	No	Yes		13.1	92	0.53	CRS with NP	CRS with NP	CRS with NP	2	1	1	1	1	1	1	1	7	1	2	7	4.5	Moderate
294	49	F	1	1	120			No	No	No	No	No	No	No		11.7	106		Normal	Anosmia	Idopathic	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
295	45	F	1	2	48			No	No	No	No	No	No	Yes		11.5	94	0.77	CRS with NP, DNS to R	CRS with NP	CRS with NP	0	0	0	0	0	0	0	0	0	1	0	0	0	Anosmia
296	34	F	3	2	36			No	No	No	No	No	Yes	No		12	126	0.77	AR	AR, Anosmia	AR	6	0	0	0	1	1	0	0	2	0	6	2	4	Moderate
297	24	F	1	2	60			No	No	No	No	No	No	No		15.8	78	0.53	B/L sphenoethmoidal polyp	B/L sphenoethmoidal polyp	SNP	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
298	59	F	2	2	36		Hypothyroid	No	No	Yes	No	No	Yes	No		12.1	95	0.85	AR, DNS to L	AR, Anosmia	AR	2	0	1	1	1	0	0	0	3	1	2	3	2.5	Severe
299	52	M	2	0				No	No	No	No	No	Yes	No		14.5	89	0.89	AR, B/L ITH	AR	AR	0	1	1	1	1	0	1	1	6	1	0	6	3	Severe
300	62	M	3	2	36			No	No	No	No	No	No	No		13.1	114	0.89	High DNS to R	Anosmia	Idopathic	1	1	1	1	1	1	1	1	7	1	1	7	4	Moderate
301	23	M	3	2	72			No	No	No	No	No	No	Yes		16.2		0.97	CRS, DNS to R	CRS	CRS without NP	2	1	1	1	1	0	1	1	6	1	2	6	4	Moderate
302	15	M	4	0				No	No	No	No	No	No	No		13.7	108	0.98	L recurrent AC polyp	L recurrent AC polyp	SNP	3	1	1	1	1	1	1	1	7	1	3	7	5	Mild
303	45	M	1	2	120			No	No	No	No	No	No	Yes		14.2	123	0.79	CRS with NP	CRS with NP	CRS with NP	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
304	47	F	1	0				No	No	No	No	No	No	Yes		12.3	123	0.67	CRS	CRS	CRS without NP	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
305	46	M	4	0				No	No	No	No	No	Yes	No		11.9	161	1.24	AR, high DNS to R	AR	AR	3	1	1	1	1	1	1	1	7	1	3	7	5	Mild
306	40	M	1	2	180			No	No	No	No	No	No	Yes		15.8	96	1.11	CRS with NP	CRS with NP	CRS with NP	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
307	46	M	2	0			DM	Yes	No	No	No	No	Yes	No		13.1	143	0.76	AR	AR	AR	0	1	1	1	1	1	1	1	7	1	0	7	3.5	Severe
308	54	M	1	1	120	RTA		No	No	No	No	No	Yes	No		15.2	145	1.21	AR	Post traumatic Anosmia	Post traumatic Anosmia	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
309	45	M	1	1	4	Fever		No	No	No	No	No	No	No		10.6	96	0.73	Spur to L	Post viral Anosmia	Post viral Anosmia	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
310	63	M	1	2				No	No	No	No	No	Yes	No		12.7	121	0.96	AR	AR, Anosmia	AR	0	1	1	0	0	0	1	0	3	1	0	3	1.5	Anosmia
311	26	M	3	0				No	No	No	No	No	Yes	No		13.6	89	0.59	AR, DNS with spur to L	AR	AR	2	1	1	0	1	1	1	1	6	1	2	6	4	Moderate
312	49	F	2	2	5			No	No	No	No	No	Yes	No					AR	AR, Anosmia	AR	0	1	1	1	0	0	1	0	4	1	0	4	2	Severe
313	25	F	1	0				No	No	No	No	No	No	Yes		12.7	85	0.2	CRS with NP	CRS with NP	CRS with NP	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
314	26	M	3	0				No	No	No	No	No	Yes	No		12.1	90	0.63	AR	AR	AR	4	1	0	0	1	1	1	1	5	1	4	5	4.5	Moderate
315	54	F	3	0			DM	Yes	No	No	No	No	Yes	No		9.7	223	1.53	AR	AR	AR	3	1	1	1	1	0	1	1	6	1	3	6	4.5	Moderate
316	41	F	1	2	7	URTI		No	No	No	No	No	Yes	No		11.9	96	0.75	AR	Post viral Anosmia	Post viral Anosmia	0	1	1	0	0	0	1	0	3	1	0	3	1.5	Anosmia
317	51	M	1	2	36		DM	Yes	No	No	No	No	No	No		12.5	149	1.32			Idopathic	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
318	40	M	1	0			Dyslipidemia, DM	Yes	No	No	No	Yes	Yes	No		15.5	93	0.92	AR	AR, Anosmia	AR	0	0	0	0	0	0	0	0	0	1	0	0	0	Anosmia
319	24	F	2	2	7			No	No	No	No	No	No	No	L COM squamosal inactive	13.8	97	0.53	DNS to R	Anosmia	Idopathic	0	1	1	1	1	0	1	0	5	1	0	5	2.5	Severe
320	47	M	3	0				No	No	No	No	No	Yes	No		14	102	1.06	AR	AR	AR	2	1	1	1	1	1	1	1	7	1	2	7	4.5	Moderate
321	49	M	1	2	60		SHTN	No	Yes	No	No	No	No	Yes		8.4	110	0.98	CRS with NP	CRS with NP	CRS with NP	0	0	0	0	0	0	0	0	0	1	0	0	0	Anosmia
322	40	F	1	2	48			No	No	No	No	No	No	No		12.9	96	0.59	DNS to R	AR, Anosmia	AR	0	1	1	0	0	0	0	0	2	1	0	2	1	Anosmia
323	54	M	1	2	84			No	No	No	No	No	No	Yes		11.3	96	0.83	CRS with NP, DNS to R	CRS with NP	CRS with NP	0	0	0	0	0	0	0	0	0	1	0	0	0	Anosmia
324	60	M	1	2	36			No	No	No	No	No	No	Yes		12.9	125	0.66	CRS with NP	CRS with NP	CRS with NP	0	0	0	0	0	0	0	0	0	1	0	0	0	Anosmia
325	45	M	1	2	6	URTI		No	No	No	No	No	Yes	No		13.6	93	0.82	AR	AR, Anosmia	AR	0	0	0	0	0	0	0	0	0	1	0	0	0	Anosmia
326	32	F	3	0				No	No	No	No	No	Yes	No		11.4	100	0.56	AR, DNS with spur to R	AR	AR	1	1	1	1	1	1	1	1	7	1	1	7	4	Moderate
327	28	M	1	2	12			No	No	No	No	No	No	No		14.9	107	0.98	Sinusitis	Sinusitis, Anosmia	CRS without NP	0	0	0	0	0	0	0	0	0	1	0	0	0	Anosmia
328	58	F	1	2	120		SHTN	No	Yes	No	No	No	No	No		14.4	107	0.53	High DNS to L	Anosmia	Idopathic	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
329	42	F	1	2	96			No	No	No	No	No	Yes	No		13.9	146	0.61	AR	AR, Anosmia	AR	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
330	59	M	4	2	120		SHTN	No	Yes	No	No	No	No	Yes		11.2	148	1.11	CRS with NP	CRS with NP	CRS with NP	3	1	1	1	1	1	1	1	7	1	3	7	5	Mild
331	66	M	1	2	36		SHTN	No	Yes	No	No	No	No	No		10.1	112	1.29	Atrophic rhinitis	Atrophic rhinitis, Anosmia	Atrophic rhinitis	0	0	0	0	0	0	0	0	0	1	0	0	0	Anosmia
332	34	M	1	2	180	RTA		No	No	No	No	No	No	No		14.1	101	0.73	DNS with spur to L	Post traumatic Anosmia	Post traumatic Anosmia	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
333	43	M	2	2	4			No	No	No	No	No	No	No				0.81	Sharp spur to L	Anosmia	Idopathic	2	0	1	1	0	0	1	0	3	0	2	3	2.5	Severe
334	61	M	4	0			DM, SHTN, IHD	Yes	Yes	No	No	No	Yes	No		12.9	142	1	AR, DNS with spur to R	AR	AR	3	1	1	1	1	1	1	1	7	1	3	7	5	Mild
335	39	M	1	2	12		DM	Yes	No	No	No	No	Yes	No		15.5	251	0.88	AR, DNS with spur to L	AR, Anosmia	AR	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
336	36	M	1	2	120		SHTN	No	Yes	No	No	No	Yes	No		15.8	97	0.73	AR	AR, Anosmia	AR	0	1	0	1	0	0	0	0	2	1	0	2	1	Anosmia
337	36	F	1	1	1	RTA		No	No	No	No	No	No	No					Normal	Post traumatic Anosmia	Post traumatic Anosmia	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
338	43	M	1	2	72			No	No	No	No	No	No	Yes		16.7	108	0.67	CRS	CRS	CRS without NP	1	0	0	0	0	0	0	0	0	1	1	0	0.5	Anosmia
339	38	F	1	2	6		DM	Yes	No	No	No	No	No	Yes		12	331	0.71	CRS with NP	CRS with NP	CRS with NP	0	0	1	0	0	0	0	0	1	0	0	1	0.5	Anosmia
340	41	M	1	2	72		SHTN	No	Yes	No	No	No	No	No		13.9	106	0.84	R SNP	SNP	SNP	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
341	55	M	3	0			DM, SHTN	Yes	Yes	No	No	No	Yes	No		12.9	86	1.26	AR	AR	AR	2	1	1	1	1	1	1	1	7	1	2	7	4.5	Moderate
342	65	F	1	2	12		DM, SHTN	Yes	Yes	No	No	No	No	Yes		12.8	99	0.87	CRS with NP	CRS with NP	CRS with NP	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
343	49	M	1	2	36			No	No	No	No	No	No	Yes		14.6	109	1.05	CRS with NP	CRS with NP	CRS with NP	1	0	0	1	0	0	0	1	2	1	1	2	1.5	Anosmia
344	23	M	4	1	36	Chicken pox		No	No	No	No	No	Yes	No		15.8	89	0.91	AR	AR	AR	4	1	0	1	1	1	1	1	6	1	4	6	5	Mild
345	38	M	3	2	18			No	No	No	No	No	No	Yes		13.9	91	0.98	CRS with NP	CRS with NP	CRS with NP	4	0	1	1		1	1	1	5	1	4	5	4.5	Moderate
346	32	F	4	0				No	No	No	No	No	No	Yes		10.9		0.77	CRS with NP	CRS with NP, AR	CRS with NP	3	1	1	1	1	1	1	1	7	1	3	7	5	Mild
347	28	M	1	2	120			No	No	No	No	No	Yes	No		13.1	101	0.82	AR, Spur to R	AR	AR	0	1	0	0	0	0	1	0	2	1	0	2	1	Anosmia
348	39	M	1	2	4		DM	Yes	No	No	No	No	Yes	No		15	194/213	0.81	DNS to R, AR	AR	AR	0	1	1	0	0	0	0	0	2	1	0	2	1	Anosmia
349	53	F	1	2	12			No	No	No	No	No	No	No		12.7	118	0.59	Normal	Anosmia	Idopathic	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
350	33	M	1	2	84			No	No	No	No	No	No	No		17	53/84	0.96	DNS to L	Anosmia	Idopathic	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
351	36	M	1	2	60		SHTN	No	Yes	No	No	No	No	Yes		15.2	123	1.27	CRS with NP	CRS with NP	CRS with NP	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
352	55	F	1	2	84			No	No	No	No	No	No	No		13.2	99/148		Normal	LPR / Anosmia	Idopathic	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
353	45	M	1	2	60			No	No	No	No	No	No	Yes	Right VC keratosis	14.8	90	1.04	CRS with NP	CRS with NP	CRS with NP	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
354	58	M	1	1	7		B/L CSOM	No	No	No	No	No	No	No	RTA preceding Anosmia	14.4	103	0.95	DNS to R	Traumatic Anosmia	Post traumatic Anosmia	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
355	28	F	1	2	3			No	No	No	No	No	No	No		13.9	93	0.64	DNS to L	Anosmia	Idopathic	0	0	0	0	0	0	0	0	0	1	0	0	0	Anosmia
356	24	M	2	2	6			No	No	No	No	No	Yes	No		15.3	103	0.99	AR	AR, Anosmia	AR	0	1	1	1	1	0	1	1	6	1	0	6	3	Severe
357	38	M	3	2	3		SHTN	No	Yes	No	No	No	No	No		11.2	108	0.86	DNS to L, B/L ETD	B/L ETD, Anosmia	Idopathic	2	1	1	1	1	0	1	1	6	1	2	6	4	Moderate
358	44	F	1	2	24			No	No	No	No	No	Yes	No		9.5	155	0.65	AR, B/L ETD	AR, B/L ETD	AR	0	1	1	0	0	0	0	0	2	1	0	2	1	Anosmia
359	55	F	1	2	72		DM, SHTN	Yes	Yes	No	No	No	Yes	No		10.9	126	0.85	AR	AR, Anosmia	AR	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
360	50	F	2	0				No	No	No	No	No	No	No							Idopathic	0	1	1	1	1	1	1	1	7	1	0	7	3.5	Severe
361	32	M	3	0				No	No	No	No	No	Yes	No		14.5	99	1.06	AR	AR	AR	3	1	1	1	1	0	1	1	6	1	3	6	4.5	Moderate
362	48	M	2	0				No	No	No	No	No	No	No		12.1	85	0.68	DNS to L, spur to R	AR, LPR, Left maxillary CRS	AR	2	1	1	1	1	0	0	1	5	1	2	5	3.5	Severe
363	55	M	1	0				No	No	No	No	No	No	Yes	Post op FESS	14.9	122	0.62	B/L CRS with NP	B/L CRS with NP	CRS with NP	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
364	62	M	1	2	48			No	No	No	No	No	No	No		14	107	0.93	High DNS to R	Anosmia	Idopathic	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
365	57	F	1	2	6	URTI	Hypothyroid, Dyslipidemia	No	No	Yes	No	Yes	No	No		13.4	107	0.86	MildDNS to R	Sinusitis, Anosmia	CRS without NP	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
366	36	F	2	0				No	No	No	No	No	No	No	Pansinusitis	13.6	86	0.7	DNS to L	Pansinusitis, Anosmia	CRS without NP	0	1	1	1	1	1	1	1	7	1	0	7	3.5	Severe
367	62	M	1	2	36		SHTN	No	Yes	No	No	No	No	No		12.7	99	0.82	High DNS to L	Anosmia	Idopathic	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
368	18	M	2	0				No	No	No	No	No	No	No		14.2	88	0.94	DNS with spur to L	AR, Anosmia	AR	1	1	1	1	1	0	1	1	6	1	1	6	3.5	Severe
369	34	M	4	0				No	No	No	No	No	Yes	No		13.7	92	1.12	AR	AR	AR	3	1	1	1	1	1	1	1	7	1	3	7	5	Mild

Sl.No.	Age	Sex	Group	Onset of loss of smell	Duration (Months)	History of prior viral illness	Comorbidities	Diabetes	Hypertension	Hypothyroidism	Bronchial asthma	Dyslipidemia	AR	CRS	Others	Hb	RBS	Creatinine	RNE	Diagnosis	Nasal Disease	Test Olfactory threshold	Odour identification test									Score			Types of olfactory dysfunction
																							Cinnamon	Asafoetida	Coffee	Tea	Pepper	Clove oil	Baby powder	Total correct	Eucalyptus (Trigeminal)	Olfactory threshold	Odour identification	Composite score	
370	48	F	1	2	12			No	No	No	No	No	No	No		13.2	89	0.65	Spur to L	Anosmia	Idopathic	0	0	0	0	0	0	0	0	0	1	0	0	0	Anosmia
371	45	F	1	2	24		DM	Yes	No	No	No	No	Yes	No		10.3	139/222		AR, DNS to L	AR, Anosmia	AR	0	0	0	0	0	0	0	0	0	1	0	0	0	Anosmia
372	37	F	2	2	120			No	No	No	No	No	Yes	No		12.3	82	0.88	AR, DNS with spur to R	AR, Anosmia	AR	0	1	1	1	1	0	1	1	6	1	0	6	3	Severe
373	15	F	1	2	84			No	No	No	No	No	No	No					Normal	Anosmia	Idopathic	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
374	36	F	1	2	120			No	No	No	No	No	No	No		13.5		0.6	Atrophic rhinitis	Atrophic rhinitis	Atrophic rhinitis	0	0	0	0	0	0	0	0	0	1	0	0	0	Anosmia
375	49	M	1	2	240			No	No	No	No	No	No	No		16.3	91	1.09	High DNS to L, spur to R	Anosmia	Idopathic	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
376	57	M	3	1	3	Fever	DM	Yes	No	No	No	No	No	No		10.9	287/355	0.86	Normal	Anosmia	Idopathic	2	1	1	1	1	1	1	1	7	1	2	7	4.5	Moderate
377	40	M	1	2	120		DM, SHTN	Yes	Yes	No	No	No	No	Yes					CRS with NP	CRS with NP	CRS with NP	0	1	0	0	0	0	1	0	2	1	0	2	1	Anosmia
378	29	M	4	2	120		SHTN	No	Yes	No	No	No	Yes	No		15.4	86	0.84	AR, DNS to R	AR, Anosmia	AR	3	1	1	1	1	1	1	1	7	1	3	7	5	Mild
379	51	F	1	2	8		SHTN	No	Yes	No	No	No	No	No		12.6	132	0.67	LPR, ETD	LPR, ETD	Idopathic	0	0	0	0	0	0	0	0	0	1	0	0	0	Anosmia
380	49	F	1	2	72			No	No	No	No	No	No	No		12.9	99	0.51	Normal	Anosmia	Idopathic	0	0	0	0	0	0	0	0	0	1	0	0	0	Anosmia
381	40	F	2	0				No	No	No	No	No	Yes	No		13.5	91	0.62	AR, B/L ETD, DNS with spur	AR, B/L ETD	AR	0	1	1	0	1	1	1	0	5	1	0	5	2.5	Severe
382	36	M	2	0			Hypothyroid	No	No	Yes	No	No	Yes	No		18.6	77	1	AR, spur to R	AR, Anosmia	AR	0	1	1	1	1	0	1	0	5	1	0	5	2.5	Severe
383	53	F	2	2	24		Hypothyroid, SHTN, CAD, BA	No	Yes	Yes	Yes	No	No	Yes		12.6	122	0.83	CRS with NP	CRS with NP	CRS with NP	0	1	1	1	1	1	1	0	6	1	0	6	3	Severe
384	55	F	2	1	12	Fever	DM, SHTN	Yes	Yes	No	No	No	Yes	No	Parosmia	11.8	153/244	0.48	AR	AR, Anosmia	AR	0	1	1	1	0	0	1	1	5	1	0	5	2.5	Severe
385	21	M	3	2	48			No	No	No	No	No	Yes	No		16.2	99	1.13	AR	AR, Anosmia	AR	1	1	1	1	1	1	1	1	7	1	1	7	4	Moderate
386	40	F	1	2				No	No	No	No	No	Yes	No		13.1	95	0.65	AR	AR, Anosmia	AR	0	0	0	0	0	0	0	0	0	1	0	0	0	Anosmia
387	48	F	0	0			BA	No	No	No	Yes	No	No	Yes		12.9	93	0.7	CRS with NP	CRS with NP	CRS with NP	5	1	1	1	1	1	1	1	7	1	5	7	6	Normosmia
388	44	M	1	2	30			No	No	No	No	No	No	No	Post op septoplasty and FESS	14.6	91	0.93	Grade 2 SNP left , multiple synechiae	Grade 2 SNP left nose	SNP	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
389	56	M	1	2	36		Dyslipidemia	No	No	No	No	Yes	No	No	Post op R hemithyroidectomy	14	156	1.39	normal	Anosmia	Idopathic	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
390	23	M	1	0				No	No	No	No	No	No	Yes		14.1	94	1.19	CRS with NP	CRS with NP	CRS with NP	1	0	0	1	1	0	0	0	2	0	1	2	1.5	Anosmia
391	30	M	1	2	12			No	No	No	No	No	Yes	No		14	89	0.82	AR	AR	AR	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
392	48	F	1	0			DM, BA	Yes	No	No	Yes	No	No	Yes		13	317/456	0.61	CRS with NP	CRS with NP	CRS with NP	0	0	0	0	0	0	0	0	0	1	0	0	0	Anosmia
393	50	F	1	2	12		SHTN	No	Yes	No	No	No	No	No		12.1	64/85	0.93	DNS with spur to R	Anosmia	Idopathic	0	0	0	0	0	0	0	0	0	1	0	0	0	Anosmia
394	35	F	2	2	2		Hypothyroid	No	No	Yes	No	No	No	Yes		13.1	94	0.82	CRS with NP	CRS with NP	CRS with NP	1	1	1	1	1	0	1	1	6	1	1	6	3.5	Severe
395	53	M	1	2	120		BA	No	No	No	Yes	No	Yes	No					AR, DNS to R	AR, Anosmia	AR	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
396	63	F	1	1	48	RTA	DM	Yes	No	No	No	No	Yes	No		11.9	43/65	0.65	AR, Left sphenoid sinusitis	AR, Sinusitis	CRS without NP	0	1	0	1	0	1	0	0	3	0	0	3	1.5	Anosmia
397	27	M	2	1	8	Fall		No	No	No	No	No	No	No		15	87	0.79	Bilateral DNS with spur to R	Anosmia	Idopathic	0	1	0	1	1	0	0	1	4	1	0	4	2	Severe
398	43	F	1	2	48			No	No	No	No	No	Yes	No		13.5	105	0.58	AR, DNS with spur to R	AR, Anosmia	AR	0	0	0	0	0	0	0	0	0	1	0	0	0	Anosmia
399	38	M	2	2	2			No	No	No	No	No	No	No					DNS to R	Anosmia	Idopathic	0	1	1	1	1	0	1	0	5	1	0	5	2.5	Severe
400	41	M	1	2	12			No	No	No	No	No	No	Yes		14.5	83	0.82	CRS with NP	CRS with NP	CRS with NP	0	0	0	0	0	0	0	0	0	1	0	0	0	Anosmia
401	36	M	1	1	1	RTA		No	No	No	No	No	Yes	No		18.5		0.88	AR, DNS to L	AR, Anosmia	AR	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
402	44	M	1	2	24		IGT	No	No	No	No	No	No	Yes		12.2	121/170	0.84	CRS with NP, DNS to L	CRS with NP	CRS with NP	0	0	0	0	0	0	0	0	0	1	0	0	0	Anosmia
403	54	F	1	2	36		SHTN	No	Yes	No	No	No	Yes	No				O.97	AR	AR, Anosmia	AR	0	0	0	0	0	0	0	0	0	1	0	0	0	Anosmia
404	43	F	2	2	72		Hypothyroid	No	No	Yes	No	No	Yes	No		11.1	102	0.56	AR, B/L ETD	AR, B/L ETD	AR	0	1	1	1	0	0	1	1	5	1	0	5	2.5	Severe
405	36	F	2	2	36			No	No	No	No	No	No	Yes		12.4	98	0.51	CRS, DNS to L	CRS	CRS without NP	1	1	1	1	0	0	1	1	5	1	1	5	3	Severe
406	61	F	1	1	6	Brain aneurysm surgery	SHTN	No	Yes	No	No	No	No	No		12	134	0.97	DNS with spur to R	Post op left ICA aneurysm Anosmia	Idopathic	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
407	33	F	4	2	6			No	No	No	No	No	Yes	No		12.5	98	0.6	AR	AR	AR	3	1	1	1	1	1	1	1	7	1	3	7	5	Mild
408	40	F	1	0				No	No	No	No	No	No	No		12.9	130	0.89	Spur to L	AR	AR	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
409	35	M	1	2	180			No	No	No	No	No	No	Yes		12.8	79		DNS to R, spur to L, CRS	CRS	CRS without NP	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
410	18	M	1	1	2	RTA		No	No	No	No	No	No	No		12.2		0.59	DNS to L	Post traumatic Anosmia	Post traumatic Anosmia	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
411	52	M	3	2	60			No	No	No	No	No	No	Yes		15.4	133	0.91	CRS with NP	CRS with NP, AR	CRS with NP	3	1	1	1	0	1	1	0	5	1	3	5	4	Moderate
412	36	M	2	2	240		DM, SHTN	Yes	Yes	No	No	No	Yes	No		12.2	113	0.82	AR	AR	AR	2	1	0	1	0	0	1	0	3	1	2	3	2.5	Severe
413	28	M	2	0				No	No	No	No	No	No	No		17.6	107	0.89	Right nasal mass	REAH/ SNP	SNP	0	1	1	1	1	1	1	1	7	1	0	7	3.5	Severe
414	34	F	4	2	2		BA	No	No	No	Yes	No	Yes	No		11.5		0.56	AR	AR	AR	3	1	1	1	1	1	1	1	7	1	3	7	5	Mild
415	40	F	1	2	36		DM	Yes	No	No	No	No	No	No		13.9	217		High DNS to L	Anosmia	Idopathic	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
416	36	F	2	2	360			No	No	No	No	No	No	No		13.1	104		DNS to R, septal spur	Anosmia	Idopathic	0	1	1	1	1	0	1	0	5	1	0	5	2.5	Severe
417	27	F	2	0				No	No	No	No	No	No	No		12.1	94	0.68	DNS to L with septal spur	Vasomotor rhinopathy	Idopathic	2	1	1	1	1	0	1	0	5	1	2	5	3.5	Severe
418	27	M	2	0				No	No	No	No	No	No	Yes		15.8	101	0.92	CRS	CRS, AR	CRS without NP	0	1	1	1	0	0	1	0	4	1	0	4	2	Severe
419	21	M	2	0				No	No	No	No	No	No	Yes		13.2	95	1.01	CRS	CRS	CRS without NP	0	1	1	1	1	1	1	1	7	1	0	7	3.5	Severe
420	29	M	2	0				No	No	No	No	No	No	No		14.3	106	0.87	DNS to L with septal spur	AR	AR	0	1	1	1	1	0	1	0	5	1	0	5	2.5	Severe
421	46	M	2	1	2	Fever		No	No	No	No	No	No	No		14.4	258	0.97	DNS to L with spur	Anosmia	Idopathic	0	1	1	0	1	0	1	1	5	1	0	5	2.5	Severe
422	52	F	3	2	240		DM	Yes	No	No	No	No	No	No	LPRD	11.7	331/411	0.77	High DNS to R	Anosmia	Idopathic	2	1	1	1	1	1	1	1	7	1	2	7	4.5	Moderate
423	37	M	1	0				No	No	No	No	No	No	Yes		11.8	106	0.79	CRS with NP	AFRS with Pansinusitis, AR	CRS with NP	0	0	0	0	0	0	0	0	0	1	0	0	0	Anosmia
424	59	M	3	0			SHTN, BA	No	Yes	No	Yes	No	Yes	No		14.9	62/103	0.91	AR, Bilateral SNP	AR, Bilateral SNP	SNP	1	1	1	1	1	1	1	1	7	1	1	7	4	Moderate
425	37	M	2	0				No	No	No	No	No	Yes	No		15	126	0.92	AR, DNS with spur to R	AR, Rhinitis medicamentosa	AR	0	1	1	1	1	1	1	0	6	1	0	6	3	Severe
426	35	F	3	2	4			No	No	No	No	No	Yes	No		13.5			AR, DNS with spur to L	AR	AR	2	1	1	1	1	1	1	1	7	1	2	7	4.5	Moderate
427	44	M	3	2	12		DM	Yes	No	No	No	No	Yes	No		15.8	101/106	1	AR	AR	AR	2	1	1	1	1	1	1	1	7	1	2	7	4.5	Moderate
428	48	M	1	0			SHTN	No	Yes	No	No	No	Yes	No		16.7	93	0.92	AR, DNS	AR	AR	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
429	29	F	3	0			DM, SHTN	Yes	Yes	No	No	No	No	No		12	116	0.91	DNS to R, spur to L, R ETD	AR	AR	2	1	1	1	1	1	1	1	7	1	2	7	4.5	Moderate
430	60	M	2	2	24		IHD	No	No	No	No	No	No	No		13.3	111	1.1	Normal	Anosmia	Idopathic	0	1	0	1	1	1	1	1	6	1	0	6	3	Severe
431	40	M	3	2	240			No	No	No	No	No	Yes	No		15	94	0.91	AR, DNS with spur to R	AR	AR	2	0	1	1	1	1	1	1	6	1	2	6	4	Moderate
432	63	F	1	1	6			No	No	No	No	No	Yes	No		12.4	95	0.71	AR	AR	AR	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
433	34	M	3	0				No	No	No	No	No	No	No		14.4	121	0.92	DNS with spur to R	Anosmia	Idopathic	2	1	1	1	1	1	1	1	7	1	2	7	4.5	Moderate
434	22	M	4	0				No	No	No	No	No	No	No		15.8	100	1.01	Bilateral high DNS	AR	AR	3	1	1	1	1	1	1	1	7	1	3	7	5	Mild
435	43	M	3	1	1	Fever	SHTN	No	Yes	No	No	No	No	No		13.3	88	0.84	DNS to L with spur	Anosmia	Idopathic	2	1	1	1	1	1	1	1	7	1	2	7	4.5	Moderate
436	30	F	1	0				No	No	No	No	No	No	No		13.4	84	0.71	Normal	AR	AR	3	0	0	0	0	0	0	0	0	0	3	0	1.5	Anosmia
437	24	M	1	1	5	RTA		No	No	No	No	No	No	No		14.8	76	0.72	DNS to R	Post traumatic Anosmia	Post traumatic Anosmia	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
438	34	M	3	2	264		BA	No	No	No	Yes	No	No	No	Multiple nasal surgeries	13.7	97	1.04	Post op status - FESS	CRS Post op	Post FESS	3	1	1	1	1	0	1	1	6	1	3	6	4.5	Moderate
439	46	M	1	0			DM	Yes	No	No	No	No	No	No		14.4	207	0.62	DNS to R	CRS with NP	CRS with NP	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
440	47	M	1	2	120		DM	Yes	No	No	No	No	Yes	No		16.6	122	0.85	AR, B/L ETD	AR	AR	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
441	64	M	2	2	72			No	No	No	No	No	No	Yes	Bilateral FESS	13.9	96	1.11	CRS, DNS to R	CRS, AR	CRS without NP	0	1	1	1	1	0	1	1	6	1	0	6	3	Severe
442	42	M	2	2	18			No	No	No	No	No	No	No		15.6		0.95	Normal	Anosmia	Idopathic	1	1	1	1	1	0	1	0	5	1	1	5	3	Severe
443	17	M	3	0			BA	No	No	No	Yes	No	Yes	No		14.9	77	0.88	AR, DNS with spur to R	AR	AR	2	1	1	1	1	1	1	1	7	1	2	7	4.5	Moderate
444	54	M	1	2	12		SHTN	No	Yes	No	No	No	Yes	No		14.8	111	1.2	AR	AR, Anosmia	AR	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
445	52	M	2	0				No	No	No	No	No	Yes	No		15	135	1.19	AR, DNS with spur to L	AR	AR	0	1	1	1	1	1	1	1	7	1	0	7	3.5	Severe
446	34	M	1	2	24			No	No	No	No	No	No	No		15.9	90	0.97	Atrophic rhinitis	Atrophic rhinitis, Anosmia	Atrophic rhinitis	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
447	21	M	1	2	48			No	No	No	No	No	No	No		15	109	0.85	Atrophic rhinitis	Atrophic rhinitis, Anosmia	Atrophic rhinitis	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
448	25	M	3	1	252		DM	Yes	No	No	No	No	Yes	No		16.6	312	0.91	AR	Post traumatic Anosmia	Post traumatic Anosmia	2	1	1	1	1	1	1	1	7	1	2	7	4.5	Moderate
449	24	M	2	0				No	No	No	No	No	No	No		15.5	83	0.83			Idopathic	0	1	1	1	1	0	1	0	5	1	0	5	2.5	Severe
450	58	M	1	0				No	No	No	No	No	No	No		15.7	119	0.79	Atrophic rhinitis	Atrophic rhinitis	Atrophic rhinitis	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
451	26	M	1	2	48			No	No	No	No	No	No	No		16.1	88	1.15	Atrophic rhinitis	Atrophic rhinitis, Anosmia	Atrophic rhinitis	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
452	51	M	4	2	12			No	No	No	No	No	No	No		14.2	110	0.89	High DNS to B/L	Anosmia	Idopathic	3	1	1	1	1	1	1	1	7	1	3	7	5	Mild
453	72	M	1	2	6			No	No	No	No	No	No	No					DNS to L	Anosmia	Idopathic	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
454	43	M	4	2	108		SHTN	No	Yes	No	No	No	Yes	No				0.98	AR	AR, Anosmia	AR	3	1	1	1	1	1	1	1	7	1	3	7	5	Mild
455	43	F	1	2	12			No	No	No	No	No	No	Yes					CRS	CRS	CRS without NP	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
456	46	M	1	2	180			No	No	No	No	No	No	Yes		13.2	96	0.9	CRS	CRS	CRS without NP	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
457	59	M	1	2	300			No	No	No	No	No	Yes	No		12.6	153	1.08	AR	AR, Anosmia	AR	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
458	52	F	3	2	48		BA	No	No	No	Yes	No	Yes	No		12.3	101		AR	AR, Anosmia	AR	2	1	1	1	1	1	1	1	7	1	2	7	4.5	Moderate
459	71	F	1	2	36			No	No	No	No	No	Yes	No		11.7	86	0.57	AR	AR, Anosmia	AR	0	0	0	0	0	0	0	0	0	1	0	0	0	Anosmia
460	33	F	1	2	60			No	No	No	No	No	No	No		12.9	126	0.59	Atrophic rhinitis	Atrophic rhinitis	Atrophic rhinitis	1	0	0	0	0	0	0	0	0	1	1	0	0.5	Anosmia
461	61	M	1	2	7			No	No	No	No	No	Yes	No					AR	AR	AR	1	0	1	0	0	0	1	0	2	1	1	2	1.5	Anosmia
462	24	M	3	0				No	No	No	No	No	No	No		14.6	88	0.99	DNS with spur to L	AR, B/L maxillary sinusitis	AR	2	1	1	1	1	1	1	1	7	1	2	7	4.5	Moderate
463	40	F	2	2	4			No	No	No	No	No	No	No		8.8	90	0.66	DNS to L	AR	AR	0	1	1	1	1	0	1	1	6	1	0	6	3	Severe
464	65	M	1	2	12		DM, SHTN, COPD	Yes	Yes	No	No	No	Yes	No		13.3	117	1.37	AR, B/L ETD	AR, Anosmia	AR	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
465	55	F	1	2	72		SHTN	No	Yes	No	No	No	Yes	No		12.1	327	0.81	AR	AR, Anosmia	AR	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
466	41	M	1	1	36	RTA		No	No	No	No	No	Yes	No		14.8	108	1.08	AR	Post traumatic Anosmia	Post traumatic Anosmia	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
467	30	M	1	2	24			No	No	No	No	No	Yes	No		14.1	85	0.7	AR	AR, Anosmia	AR	0	0	0	0	0	0	1	0	1	1	0	1	0.5	Anosmia
468	31	F	1	1	1	URTI, Fever		No	No	No	No	No	Yes	No		14			AR	Post viral Anosmia	Post viral Anosmia	0	0	1	0	0	0	0	0	1	1	0	1	0.5	Anosmia
469	38	M	1	2	120		Hepatitis B	No	No	No	No	No	Yes	No		16.2		1	AR, B/L ETD, Spur to L	AR, Anosmia, Hep B	AR	0	0	1	0	0	0	1	0	2	1	0	2	1	Anosmia
470	37	M	3	2	48		BA	No	No	No	Yes	No	No	No		13.5	95	0.95	DNS with spur to L	AR	AR	2	1	1	1	1	1	1	1	7	1	2	7	4.5	Moderate
471	35	F	4	0				No	No	No	No	No	No	Yes		12.2	78	0.62	DNS with spur to R, CRS with NP	CRS with NP	CRS with NP	3	1	1	1	1	1	1	1	7	1	3	7	5	Mild
472	39	M	3	0			SHTN	No	Yes	No	No	No	Yes	No		12.9	105	1.12	AR, DNS to L	AR	AR	2	1	1	1	1	1	1	1	7	1	2	7	4.5	Moderate
473	44	M	1	2	120			No	No	No	No	No	No	No	Post op B/L Youngs	16.4	92		Post op status - Youngs	Atrophic rhinitis	Atrophic rhinitis	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
474	57	F	2	0			SHTN	No	Yes	No	No	No	No	No		11.5		0.61	DNS with spur to L	Vasomotor rhinitis	Idopathic	2	1	0	0	1	1	1	1	5	0	2	5	3.5	Severe
475	61	M	1	2	240			No	No	No	No	No	No	Yes		14.8	100	0.97	CRS with NP	CRS with NP	CRS with NP	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
476	48	M	2	2	24			No	No	No	No	No	No	No		13.2	83	0.84	DNS with spur to R	Anosmia	Idopathic	0	1	1	1	1	1	1	0	6	1	0	6	3	Severe
477	32	F	1	1	3	RTA		No	No	No	No	No	Yes	No		11.5		0.55	AR, DNS to R	Post traumatic Anosmia	Post traumatic Anosmia	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
478	37	F	3	0				No	No	No	No	No	No	Yes		12.9	113	0.57	CRS with NP	CRS with NP	CRS with NP	2	1	1	1	1	1	1	1	7	1	2	7	4.5	Moderate
479	34	M	2	0				No	No	No	No	No	No	No							Idopathic	0	1	0	1	1	0	1	0	4	1	0	4	2	Severe
480	41	F	4	0				No	No	No	No	No	Yes	No		14	87		AR	AR	AR	3	1	1	1	1	1	1	1	7	1	3	7	5	Mild
481	47	F	2	0			Hypothyroid	No	No	Yes	No	No	No	Yes		10.2	91	0.74	CRS with NP, DNS to L	CRS with NP	CRS with NP	0	1	1	1	1	0	1	1	6	1	0	6	3	Severe
482	51	M	1	1	18	Fall		No	No	No	No	No	Yes	No		12.7		0.89	AR	Post traumatic Anosmia	Post traumatic Anosmia	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
483	14	F	4	0				No	No	No	No	No	No	No	Right frontoethmoidal mucocele	12.4	100	0.67	Right mucocele	Right mucocele	Post FESS	3	1	1	1	1	1	1	1	7	1	3	7	5	Mild
484	33	M	4	2	3			No	No	No	No	No	No	No					DNS to R	Anosmia	Idopathic	3	1	1	1	1	1	1	1	7	1	3	7	5	Mild
485	36	M	4	2	240			No	No	No	No	No	No	Yes		15.2	111	1.05	CRS with NP	CRS with NP	CRS with NP	5	1	1	1	1	0	1	1	6	1	5	6	5.5	Mild
486	43	M	0	0			Hypothyroid	No	No	Yes	No	No	No	Yes		14.4	123	0.84	CRS with NP	CRS with NP	CRS with NP	6	1	1	1	1	1	1	1	7	1	6	7	6.5	Normosmia
487	47	F	4	2	12			No	No	No	No	No	No	No		12	90	0.64	DNS with spur to L	Anosmia	Idopathic	3	1	1	1	1	1	1	1	7	1	3	7	5	Mild
488	42	M	0	2	60			No	No	No	No	No	No	Yes		16.1	82	1.22	CRS with NP	CRS with NP	CRS with NP	5	1	1	1	1	1	1	1	7	1	5	7	6	Normosmia
489	40	F	1	1	1	Fever		No	No	No	No	No	No	No		13.7	206	0.76	Normal	Post viral Anosmia	Post viral Anosmia	0	0	0	0	0	0	0	0	0	0	0	0	0	Anosmia
490	44	M	3	0				No	No	No	No	No	Yes	No		12.4	119	0.78	AR	AR	AR	1	1	1	1	1	1	1	1	7	1	1	7	4	Moderate